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PRIMARY CHRONIC SPLENOMEGALY*

Banti's Disease

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THE complicated group of the so-called primary splenomegalies has been considerably simplified as a result of recent investigations. Thus Gaucher's disease, which was regarded by its original author as a primary epithelioma of the spleen, and more recently as an endothelioma or a cryptogenetic splenomegaly, is at present looked upon as a disturbance in the metabolism of certain lipoid substances in which the entire body (the Makrophage System) is involved. The spleen is, therefore, not more involved in the process than the liver, the bone marrow, and other organs or structures. Moreover, the disease has been lately dissociated into two distinct entities: the "Gaucher"[1] and the "Niemann-Pick" diseases[2].

Likewise with the progress in hematology a number of other instances of splenic enlargement formerly regarded as primary are today interpreted as secondary, being merely one link in the chain of disturbances in the hemato-lympho-myelopoietic systems.

There is one group of splenomegalies still regarded as primary, which is of interest in that the early removal of the spleen results in many instances in a cure of the disease. These splenomegalies, too, are said to be followed or accompanied by a disturbance in the liver (cirrhosis). In a number of cases, however, the spleen is found to be the sole organ that is involved. This variety of splenomegalies, of which Banti's disease[3] is an example, is included in the group of sclerogenic disorders.

It would appear that even these splenomegalies are probably not all idiopathic, but are the result of extrinsic influences which I shall discuss in the comment on the case to be reported.

A boy of ten with a history of profuse hemorrhages of six years' duration. Clinical finding of an enlarged spleen and a hypochromic anemia. Transfusions. Death. Necropsy disclosure of splenomegaly (type Banti).

History: (B. I. H. No. 5211). M. J., a Jewish boy, aged ten, was admitted to the Beth Israel Hospital with the complaint of vomiting of blood. His father and mother were living and in good health. His mother had one miscarriage two years prior to the boy's admission to the hospital, after which there were no other pregnancies. There was no history in the family of syphilis, tuberculosis, malaria or degenerative or metabolic diseases.

When the mother was in her third month of pregnancy with this child, sugar was found in her urine which persisted up to the delivery. At the eighth month of pregnancy she developed influenza and also a condition which was diagnosed by the local physician as "Bright's disease." Labor was induced by high forceps resulting in the delivery of the boy who appeared to be normally developed, weighing only 4.5 pounds (about 2 kg.). However, he gained weight rapidly. About two weeks following birth he developed an abscess on the left leg which persisted for three months. He was breast-fed for two weeks only. Teeth appeared at the sixth month. He did not walk until the age of three because of a left hemiplegia which he developed at the age of one and a half years, and which probably resulted from a poliomyelitis. He had measles at the age of two years, whooping cough at three years, and mumps at the time of present admission. His tonsils and adenoids were removed six years prior to his admission. He was vaccinated on three occasions with negative results. The Schick test was negative.

At the age of four, on the day following tonsillectomy, he vomited bright red blood which was thought to be "postoperative". Similar bleedings occurred on three more occasions during the next two days, and also one month later, at which time he vomited about one quart of bright red blood. He also was complaining of pain in the abdomen. He was then referred to the Children's Hospital, Boston, where, following a hemorrhage, he was transfused. He remained in the hospital nine weeks, and was discharged improved. The diagnoses at the Children's Hospital were bronchopneumonia, secondary anemia and septicemia.

He was in good health until about three years prior to the admission to the Beth Israel Hospital, when he again had two hemorrhages, vomiting about one quart and one pint respectively of bright red blood, within an interval of six hours. He was then referred to a hospital where, following a hemorrhage, he was transfused. He rapidly improved and was discharged improved two weeks later. He again was well for about two years, when suddenly he developed pain in the abdomen and nausea followed by the vomiting of about one pint of "coffee ground" material which contained food. For the last year before his entry to the Beth Israel Hospital he was quite comfortable. One week before admission he developed mumps and a few days later he was complaining of severe "girdle" pain in the abd nen. He showed tarry stools. He vomited food and red blood respectively. Following this last vomiti..g attack he was referred to this hospital.

Physical examination: The boy was well developed and nourished. The skin was tan-colored, but the sclerae were clear. The head was somewhat egg-shaped. A small walnut-sized gland was palpable under the angle of the left jaw. No glands could

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be felt elsewhere. The heart, except for a loud systolic murmur, was negative, and the lungs were clear.

The spleen was palpable 3.5 cm. below the costal margin in the midaxillary line; its edge was rounded and its surface was firm and regular. The liver could not be felt. The other abdominal and pelvic viscera showed no pathologic changes. The genitourinary tract, the bones and the muscles showed nothing remarkable.

Laboratory findings: The Wassermann and the Kahn tests were negative. The erythrocyte count totaled 1,720,000 per cubic millimeter: the white cells numbered 10,600 per cubic millimeter; the platelets averaged between 120,000 and 140,000 per cubic millimeter; the reticulocytes numbered 2.6 per cent: the hemoglobin was 40 per cent. There was a moderate anisocytosis and poikilocytosis, and a slight diffuse polychromatophilia. The icteric index was 9.6. The bleeding and clotting time and the fragility test were within normal limits. The blood culture, the Widal test, and the Pirquet tuberculin test were negative. The stools showed the presence of occult blood with the benzidin reagent.

Roentgen-ray examination showed no abnormalities in the lungs; the heart was enlarged. In the bones there was some atrophy of the left tibia and also of the muscles of the left lower leg.

Course in hospital: The temperature ranged between 99.0 and 101.2 F. the first eleven days, and the pulse was around 120 per minute. The patient was transfused with 430 cc. of citrated blood, resulting in a considerable improvement of his general condition. About three weeks following transfusion he suddenly developed a severe headache, and the following day vomited about one quart of bright red blood. This recurred several times in smaller amounts. He then was transfused with 550 cc. of citrated blood. However, hematemesis recurred. Another transfusion of 450 cc. of citrated blood was done, resulting in diminution of the hematemeses which have become smaller and less frequent. The day following the last transfusion he became comatose and died after a stay of five weeks in the hospital.

It was obvious that the patient was not ill with a hemolytic anemia, thrombocytopenic affection, or a polycythemia vera. Gaucher's disease was likewise dismissed on the grounds that only the spleen appeared to be involved, whereas Gaucher's splenomegaly, as referred to, is a generalized disease affecting practically all organs and tissues of the body. The patient's age and again the nature of his blood were against his having pernicious anemia. Similarly it was determined that he did not have malaria, syphilis, or a parasitic disease; and a thorough study for the presence of a pericardial pseudocirrhosis (Friedel Pick's syndrome) which is often accompanied by a large spleen, yielded negative results. Greater difficulties were encountered in finding out whether the patient has had an aleukemic myelosis (aleukemic myelogenous leukemia). This could have been settled by a biopsy of the bone marrow, which was not done. However, the profuse hemorrhages from which the boy was suffering and the long duration of the illness (at least six years) were against this diagnosis.

Tumors that arise primarily in the spleen, such as hemangio-endothelioma or sarcoma, cause death of young patients quite rapidly, and this probably holds true also of Hodgkin's disease, which sometimes has a tendency to be confined predominantly to the spleen. It was finally thought that the disease was either an atrophic cirrhosis of the liver, which not infrequently is accompanied by a large spleen, or else Banti's disease.

Necropsy: This was performed by Dr. M. J. Schlesinger. The body was well nourished, but somewhat dehydrated. The pleural cavities contained each about 60 cc. of hemorrhagic fluid. The right lung was slightly adherent at the apex and the left was strongly attached at the base to the diaphragm. The thymus was rather large. The heart showed thickening of the walls of the left ventricle, and the lungs showed bronchopneumonia. The esophageal veins were large and thickened.

The liver weighed 690 gm. and except for paleness was not remarkable. The spleen weighed 275 gm. It was grayish-yellow and very firm. The cut surface showed a very large amount of fibrous tissue and practically no blood. The organ was not adherent to the surrounding viscera, and its surface was smooth and regular. No ruptured vessels or bleeding points and no obstruction were found in the abdominal veins. The lesser omentum was very thick, matted together, and fibrous. There was no gross evidence of obstruction of the portal vein, or in the vascular supply around the celiac axis. The left hemisphere of the brain was smaller than the right. Cultures from the blood were negative.

Microscopic examination: The lungs showed edema, congestion and patches of bronchopneumonia. Some of the smaller pulmonary vessels contained thrombi which were of recent origin; in some of them the wall was infiltrated with lymphocytes. Branches of the pulmonary artery accompanying large bronchi showed areas of intimal thickening (ancient organized thrombi) made up of dense acellular connective tissue (Fig. 1). In some

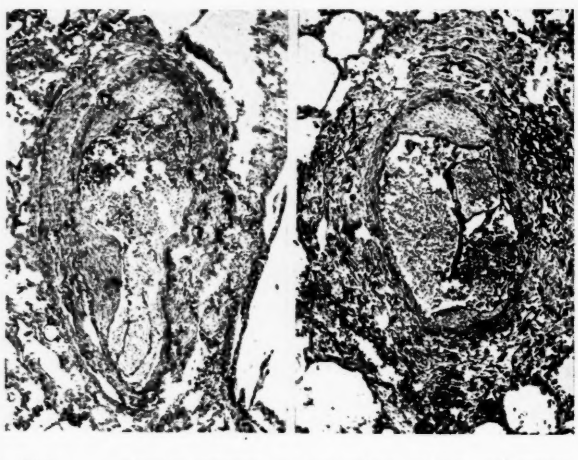

FIG. 1. Sclerotic changes in the intima of the pulmonary vessels resulting from the organization of thrombi. Methylene blue and eosin × 60.

vessels these sclerotic changes had led to a marked narrowing of the vascular lumen, and in others the newly-formed tissue showed formation of minute canaliculi (Fig. 1). The thin-walled veins also showed slight sclerotic changes of their intima. However, the septal capillaries did not seem to reveal pathologic changes. A medium-sized vein was found to be surrounded by a fairly wide collar of lymphocytes, mingled with a couple of Langhans-like

giant cells. The bronchi revealed no disease. The perivascular lymphatics were very markedly dilated and contained coagulated serum.

The spleen showed diffuse fibrosis of about two-thirds of the organ. In some areas the fibrous tissue contained fair numbers of lymphoid and reticulum cells, while in others the number of the small round cells was very limited.

The sinuses were narrow and bloodless, their lining cells showing swelling and proliferation, and their lumens containing free macrophages (Fig. 2). The follicles, too, showed fibrosis. The earlier

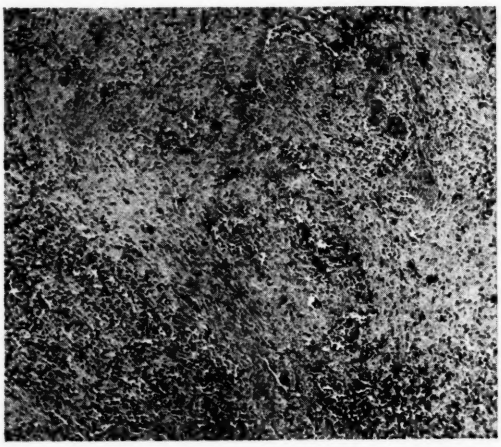

FIG. 2. Fibrosis of the spleen (Fibroadenia). The narrow and bloodless sinuses show proliferation and swelling of the cells which line their walls. Methylene blue and eosin × 95.

period of fibrous transformation (fibroadenia) is seen in Fig. 3. Here the central artery is patent, being

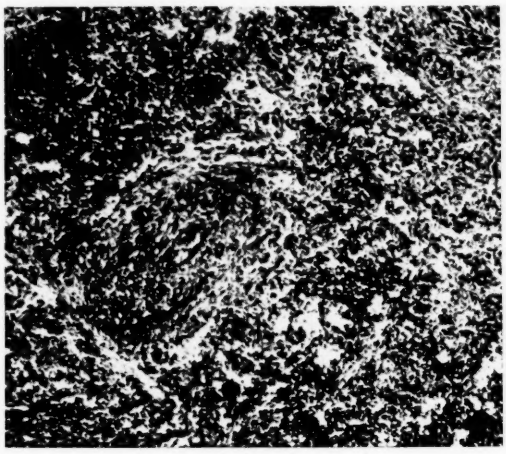

FIG. 3. The early stage of fibrous transformation of the lymph follicles in the spleen. Methylene blue and eosin × 150.

surrounded by concentric layers of connective tissue fibers and lymphoid cells. Complete fibrosis of the Malpighian body is illustrated in Fig. 2.

Parts of the spleen free from fibrosis showed the reticulum to be rather dense and the sinuses to be dilated and filled with blood, hypertrophic trabeculae and manifold thickened ellipsoids. The capsule showed dense fibrosis and the veins sclerotic changes.

The liver and the other abdominal viscera were not remarkable except for the lesser omentum, which showed fibrosis. The esophageal veins showed marked varicosities (Fig. 4).

COMMENT

The Nature of the Splenomegaly. About four decades ago the Italian pathologist Banti[3] "isolated" a morbid condition of the spleen which, as he thought, originated primarily in this organ. The cases which he had the opportunity of studying revealed to him that the disease was characterized by its clinical course and also by the pathologic changes found postmortem. Thus clinically he arbitrarily discriminated three stages of the malady. The first period, lasting from three to five years, is characterized by a large, firm, smooth and painless spleen, by a secondary (hypochromic) anemia, and by gastrointestinal symptoms. In the second period, lasting from one to two years, in addition to the enumerated signs, there also occurs a noticeable

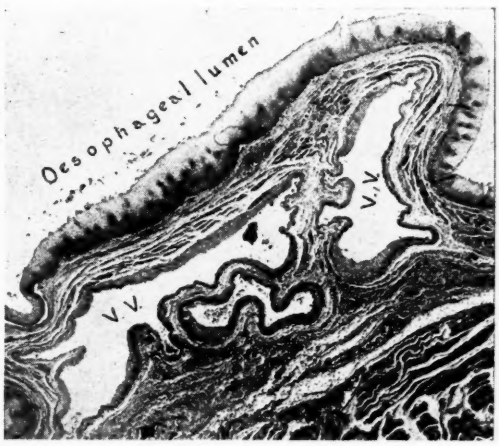

FIG. 4. Varicose veins (VV) in the wall of the esophagus. Methylene blue and eosin × 20.

enlargement of the liver, changes in the urine, and finally signs of jaundice. In the third, terminal period the liver decreases in size and ascites occurs.

At the postmortem examination, the spleen reveals changes which, according to Banti, are specific in that they begin in the follicle with a thickening and an increase in its fibers, the changes starting around the central arteries, from where they spread centrifugally (Fig. 3). Fibrosis likewise occurs in the pulp, which eventually leads to sclerosis of the organ (Fig. 2). It is interesting that, in spite of the diffuse scarring, the "adenoid" features of the spleen can be easily identified, which led Banti to designate this pathologic process as "Fibroadenia." The splenic sinuses are narrow, showing their lining cells in a state of proliferation (Fig. 2). There is no increase in blood pigment in the spleen and no hemosiderin in the liver; that is, there is no blood destruction. The bone marrow is red, which is characteristic of a secondary (hypochromic) anemia. Banti also stressed the sclerotic changes in the portal, splenic, and mesenteric veins found in his patients. He likewise emphasized that one of the prerequisites of the

disease is the fact that its etiology is unknown. In the liver he found atrophic (Laennec's) cirrhosis.

The blood, as noted, reveals anemia of the hypochromic type. There occurs poikilocytosis, anisocytosis, and often a relative and absolute mononucleosis. As a rule there is a leukopenia, but in instances when the white cell count is elevated, the leukocytosis concerns the neutrophilic elements.

Following Banti's original report, the disease bearing his name was readily accepted by clinicians and, to a lesser degree, by pathologists. However, Eppinger[4] in his authoritative monograph on "spleno-hepatic" diseases stated that none of the cases that have been diagnosed as Banti's disease up to 1915 complied with the requirements as set forth by Banti. Similar opinions were recently expressed by Hirschfeld[5] and by Lubarsch[6]. It is interesting that Osler, who in 1900 wrote[7], "I have no personal knowledge of the interesting condition described by Banti," two years later stated[8] that "a special malady does exist, of unknown etiology, characterized by a chronic course, enlargement of the spleen, anemia of a secondary type, a marked tendency to hemorrhage from the stomach, and a liability in the late stages to be associated with jaundice, cirrhosis of the liver and ascites. The conditions described as primitive splenomegaly and Banti's disease are initial and terminal stages, respectively, of this malady." Osler's cases, however, should not be included in the category of "Banti", because most of his patients had had malaria or syphilis previous to their then present illness. Again, the removed spleens were not examined histologically. In fact, two spleens from his patients, recently reinvestigated by Cushing and MacCallum[9], showed conditions unlike those found in Banti's disease.

As a result of the indiscriminating diagnoses, the entity bearing Banti's name soon became discredited. Clinicians then began to speak of "Banti's syndrome" and not of the disease, while pathologists have become skeptical as to the very existence of this malady.

The case discussed here is identified as "Banti's disease" on the basis of the clinical picture, splenomegaly of unknown etiology, hypochromic anemia and hemorrhages, and on the postmortem findings. The histologic picture of the patient's spleen described above is an exact counterpart of the picture as given originally by Banti[3], and later by Eppinger[4], Hirschfeld[5], Lubarsch[6], and Dürr[10]. The last-mentioned author had the opportunity of comparing his material with specimens obtained from Banti himself.

Pathogenesis and Specificity of the Splenic Changes. As referred to, the pathology of "Banti spleen" is characterized by a "fibroadenia", by narrow, slit-like sinuses lined by proliferated swollen cells resembling cuboidal epithelium, and finally by the absence in the spleen and liver of signs of local hemolysis (blood destruction). The statements made by pathologists to the effect that similar pictures are found in other inflammatory and congestive processes is not borne out by close observation. No one has ever demonstrated "fibroadenia" in passive congestion of the spleen of cardiac origin, or in splenomegaly which accompanies atrophic cirrhosis of the liver. Lubarsch, who is an authority on the subject, wrote[6] that "it cannot be denied that the Banti spleen shows peculiarities in the follicle and in the sinuses that cannot be found in other splenohepatic diseases".

There is, however, a disease in which the changes found in the spleen closely imitate those described by Banti. The condition which was reported under the name of "thrombotic obliteration and cavernous transformation of the portal vein," was recently the subject of a detailed study by Klemperer[11], who reported one case of this disease and reviewed twenty-two cases from the available literature. This term, according to Klemperer, has been applied to a pathologic process of the portal vein and the lesser omentum, whereby these structures present a peculiar spongy appearance. This is due to the fact that numerous fine blood channels, instead of the portal vein, penetrate the thickened ligament. In Klemperer's case it concerned a woman of 42 who was complaining of pain in the left upper abdominal quadrant. The duration of the disease was at least nine years, and the main symptoms were compatible with Banti's disease. The removed spleen showed fibroadenia, and the portal vein revealed cavernous transformation. There was no cirrhosis of the liver. Klemperer related that most of the patients with this disease (whose histories he has carefully reviewed) died as a result of thrombosis of the superior mesenteric vein, with subsequent hemorrhagic infarction and necrosis of the intestines; some died of a hemorrhage from dilated esophageal veins, and in two there occurred a peritonitis due to infection of the ascitic fluid. "The splenomegaly," affirmed Klemperer, "with the morphologic picture apparently typical of Banti's disease was the result of the previous thrombosis in the portal system."

Of great interest, too, is the report made by Wohlwill[12], who found a splenomegaly of Banti's type in a patient who showed at necropsy a *sclerosis* of the portal vein. Wohlwill's patient was a man of 59, an addict to alcohol, who was operated upon in the hepatic region of the portal vein thirty-two years previously. Death occurred as a result of a profuse intestinal hemorrhage. The necropsy revealed sclerosis of the portal vein and its branches, varicose dilatation of the inferior mesenteric vein, cirrhosis in the area of the portal vein and a voluminous spleen

with the histologic picture characteristic of Banti's disease.

Like Klemperer, Wohlwill was of the opinion that the disease in the liver and the spleen resulted from the sclerotic changes in the portal circulatory apparatus.

Banti, in his numerous articles, stressed the fact that sclerosis of the splenic and the mesenteric veins was a common finding in patients with the disease which he has identified. He was, however, of the opinion that the vascular changes *followed* those of the spleen. He expressed the hypothesis that the disease (sclerosed) spleen elaborates peculiar toxins ("miasma imaginaria", as Osler ironically put it) which pass *via* the splenic vein toward the liver, causing sclerosis of the splenic vein and ultimately cirrhosis (sclerosis) of the liver. This, however, was regarded as being improbable because of the fact that the spleen, which shows an advanced sclerosis and whose functions are therefore greatly impaired, is not in a position to be the source of a hypothetic toxin causing atrophic cirrhosis of the liver and sclerosis of the splenic and other veins[13].

There are two possible explanations for the sclerosis of the spleen and its efferent vessel: (1) That an unknown factor having a predilection for these particular structures has caused their sclerosis. It is widely known that poisons, toxins, and microörganisms oftentimes possess affinities for certain organs or structures. (2) That the hypothetic factor has primarily led to sclerosis of the splenic (and possibly also the portal and mesenteric) vein, this in its turn leading to the peculiar metamorphosis of the spleen and possibly, at a later date, to cirrhosis of the liver.

Sclerosis of veins (phlebosclerosis) as evidenced by atheromatous thickening of the wall and calcareous deposits in the intima, was described by a number of pathologists of the past century. According to Borrmann[14], these vascular lesions were known to Bichat and to Duroziez, who referred to them as to angiosclerosis. Ziegler[15] discriminated diffuse and circumscribed sclerotic lesions of the veins, and Simmonds[16] wrote of systemic diseases of the veins (systematisierten Venenerkrankungen). Of more recent writers phlebosclerosis was stressed by Wohlwill[12], and by Wilhelm Schilling[17].

The findings of Klemperer and particularly those of Wohlwill seem to me to be in favor of the hypothesis that the condition originally described by Banti is primarily a sclerosis of the veins of the portal system, and only secondarily a splenomegaly caused by a stasis protracted over a number of years, and also possibly by metabolic disturbances in the spleen resulting from its deficient circulation. It is true that fibroadenia was not observed in passive congestive processes of the spleen, but the duration of Banti's malady is usually a matter of decades, this fact making futile comparisons between this condition and acutely or subacutely stagnant spleens. Then, too, one must discriminate between stasis resulting from a general condition and that caused by a local disturbance. I, moreover, believe that the sinuses in Banti's spleen, which resemble "cystic structures lined by epithelium" and which are regarded as being "specific" are changes due to the mere shutting off of the circulation resulting from the widespread fibrosis of the organ. The pulmonary alveoli, lined by cells which are genetically related to those lining the sinuses of the spleen, display quite similar traits in sclerotic processes of the lung when the respiratory function of the air sacs is interfered with.

Finally, it is superfluous to look for signs of blood destruction in the sclerosed and practically functionless spleen in this disease. It will be remembered that in no instance was the disease investigated in its earlier stages and the pathologic process, found in the spleen of my patient, as in those reported by previous writers, is the end product of a malady which ran a course of a great number of years.

The problem of cirrhosis of the liver, which is said to follow Banti's splenomegaly, was discussed by many writers. Here, too, it is likely that the hepatic sclerosis resulted not from the "imaginary" splenotoxins of the fibrosed spleen, but was caused by the same factor that had damaged the spleen or resulted from the long-standing sclerotic changes of the entire portal circulatory mechanism or of a part of it.

The theories of the pathogenesis of thrombotic obliteration and cavernous transformation of the portal vein has been fully reviewed by Klemperer[11]. The etiology of sclerosis of the mesenteric and splenic veins is probably related to sclero-thrombotic changes in the portal system, these possibly being caused by syphilis, trauma (operative), or possibly being congenital vascular defects. According to Wohlwill, they are by far more common in younger individuals. The young patient discussed here, aside from the usual children's exanthemata, had had infantile paralysis when 1½ years old and an abscess of the leg closely following his birth, which lasted several months. The vascular changes found in the lungs (Fig. 1) have most likely resulted from this infection. The omentum too, showed peculiar fibrotic changes of the parenchyma and of some of the blood vessels. Unfortunately no tissue was available for the study of the splenic vein.

Diagnosis: The diagnosis of Banti's disease is made, first of all, by exclusion. But even after a thorough investigation it will hardly be possible to ascertain whether one is not dealing with a "banal" splenomegaly that is liable to accompany Laennec's cirrhosis. That the spleen is oftentimes greatly enlarged in portal cirrhosis

has been reported by numerous observers[18], who sometimes expressed the belief that the disease started here and not in the liver. Lubarsch[6] found that of 75 cases of annular cirrhosis of the liver the spleen weighed more than 500 gm. in 18 instances, and more than 1,000 gm. in four.

One young patient (aged 20) with portal cirrhosis, recently observed by me showed at necropsy a liver which weighed 1200 gm. and a spleen of 995 gm.

According to Snell[19], in primary portal cirrhosis an enlarged spleen occurs late, when the portal circulation is greatly restricted, and the interference with the flow of portal blood must exist for some time before collateral channels are established. However, Gilbert and Lereboullet[20] stated that "both the liver and the spleen receive blood from the arterial trunk where the hepatic and splenic arteries arise (the celiac axis) and anything that is spreading by way of the arteries is liable to affect simultaneously both organs."

I have observed that in the aged the early stages of portal cirrhosis are very often accompanied by marked enlargement of the spleen, while in the terminal period the spleen is atrophic. In youthful patients, in whom the disease runs a rather rapid course, splenomegaly is usually conspicuous throughout the entire clinical course of the malady.

Not only the splenomegaly, but also the hemorrhages are not infrequently met with in atrophic cirrhosis. It is interesting that "cirrhotic" patients with ascites are less liable to "bleed" than those whose abdominal cavities show no free fluid. This is explained on the supposition that the fluid which exudes from the vessels into the peritoneal cavity relieves the "portal hypertension".

The occurrence of hemorrhages in sclerogenic splenopathies was stressed by Osler[8] in his early reports on splenic anemia. "The vasa brevia," noted Osler, "passing from the fundus, which empty into the splenic vein, drain a large section of the stomach. From estimates of Mall and Kraus, 41 per cent of the blood of the splenic artery goes to the stomach. One may reasonably conclude, therefore, that a similar percentage of blood in the splenic vein is derived from that organ. The amount of blood brought may be enormous, and the patient may be rendered exsanguine." In many of Osler's patients, an ulcer of the stomach was found.

The question was recently reinvestigated by Smith and Howard[21] and by Grègoire and Weil[22]. The French writers emphasize the instantaneous character of the hemorrhage and the abundance of the blood that occurs in splenomegalies. In the patient presented here, the hemorrhages which occurred at intervals over a period of six years resulted from varicose veins of the stomach and the esophagus (Fig. 4).

It may be seen that this sign, too, is proper to annular cirrhosis as well as to "primitive" splenomegaly.

The picture of the peripheral blood in Banti's disease, as seen in dry films, has not been regarded as being characteristic until Rosenthal's[23] report to the effect that Banti's disease is associated with marked disturbance in the number and function of platelets. Accordingly, he discriminated: (1) A thrombocythenic group in which the platelets rise promptly (after splenectomy) and remain elevated permanently, reaching in instances the number of 800,000; (2) A thrombocytopenic group in which splenectomy is immediately followed by a gradual increase in the number of platelets. In the experience of Rosenthal this discrimination is of significance in that favorable surgical results were obtained in the second group only. This observation needs further study.

Treatment: Of the clinical triad of symptoms,—the splenomegaly, the hemorrhages, and the hypochromic anemia—the profuse bleeding is by far the most dramatic feature with which the clinician is faced. This symptom does not result from the perverted function of the spleen, but is due to the fact that the sclerosed splenic vein is opposed to the proper emptying by the spleen of its blood. The spleen, then, in its attempts to get rid of the extra blood recurs to vicarious circulation through the gastric, esophageal, and sometimes even nasal and gingival veins. Thus the veins become dilated, their walls thinned out, and their resistance weakened. They bleed readily, and as noted, are liable to cause alarming hemorrhages.

The fact that hemorrhages did not always cease following splenectomy was regarded by some observers as an evidence that the disease is primarily a general condition, the removal of the spleen being, therefore, of no avail. However, an early removal of the spleen will very likely be followed by a cure of the disease in most instances. Only the advanced patients with a well-developed vicarious circulatory mechanism will continue to suffer from hemorrhages which may be fatal even though the spleen has been removed. Here, as elsewhere, it is essential to recognize and to treat the disease in its early stages.

Summary: A case of a boy, ten years old, is reported, who showed a splenomegaly of unknown origin, a secondary anemia, and hemorrhages of six years' duration. The postmortem examination revealed a large, fibrosed spleen, with the histologic traits characteristic of Banti's disease. There was no cirrhosis of the liver. The opinion is expressed that Banti's splenomegaly is probably a primary disease of the veins of the portal circulation, and particularly that of the spleen (phlebosclerosis or phlebothrombosis of the vena splenica). The possible etiology of this condition is discussed, and

the difficulties encountered in the diagnosis of the disease are outlined.

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RESULTS AND INTERPRETATION OF A COMPLEMENT FIXATION TEST FOR GONORRHEA

BY CHANNING S. SWAN, M.D.*

DURING the past year I have taken blood for a complement fixation test for gonorrhea on three groups of private patients. The first group included those who came for treatment of a deep genital infection in whom there was no history of gonorrhea and no evidence of gonococci by smear. The second group contained those in whom there was a history of an old gonorrhea, but who now had no evidence of gonococci by smear in their present flare-up or chronic state. The third group was made up of a few of those whom I have treated from the beginning of their acute gonorrhea. The test was not employed on early acute cases in either sex as the result invariably would have been "negative". It also was not used on any chronic case which was quite recently a positive anterior case as long as there was a considerable amount of pus in the prostatic smears. Its use, therefore, was limited to doubtful cases rather than making it routine.

There were forty-four such cases. The blood in thirty-three of these was "negative", in three it was "doubtful", and in eight it was "positive". Considering the results on the basis of the groups mentioned above, it was found that of those male patients who came with a deep genital infection, with or without discharge, and in whom there was no history of gonorrhea and with urethral and prostatic smears negative for gonococci, there were thirteen "negatives" and no "positives". I believe it is safe to say that all these cases represented non-specific infections. There were also, however, three female patients with chronic endocervicitis and no history of gonorrhea, who had "negative" bloods but on whom positive smears were finally obtained after several visits.

In the group where there was a past history of gonorrhea, usually symptomatically cured, but no positive evidence by smear of the disease at the time they were first seen there were ten "negative" tests and all the eight "positive" tests. These will be discussed more fully later on.

In the third group, representing patients under care from the onset of their acute gonorrhea there were seven who were willing to continue treatment until all clinical evidence of the disease had been eradicated. All seven had "negative" complement fixation tests when discharged. Too many others deserted when symptomatically cured, free of discharge, and with all smears negative for gonococci, but with more pus in their prostates than seemed to me to warrant doing the blood test which was reserved in these cases as one of the final criteria of cure.

There were three "doubtful" bloods. These were not repeated because in each instance sufficient clinical evidence subsequently came to light to show that they should all be considered "positive". This substantiates the view of the laboratory that a "doubtful" blood has a "positive" interpretation.

The series includes eight female patients. There was one "positive" blood in a female patient who had a history of gonorrheal salpingitis which she had acquired from her husband. The husband had a positive smear from the

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urethra, but the gonococcus was not found in the wife. After her endocervicitis had been cleaned up the blood remained "positive", undoubtedly due to the old salpingitis. The other seven females had chronic endocervicitis and although all their bloods were "negative", in three of the cases as already mentioned the gonococcus was finally identified after repeated smears. It would appear from this evidence that gonorrheal endocervicitis frequently gives a "negative" complement fixation test, but if the disease has extended to the tubes a "positive" reaction may occur. On the contrary, McNeil and Barringer find "positive" reactions with the disease in the cervix or Bartholin's glands.

The seven "positive" bloods in male patients who had a history of an old gonorrhea and who came with a new flare-up but with negative smears are interesting, because some show definitely the value the test can have. Two such histories are reviewed briefly:

CASE 1. R. J. N. Had acute gonorrhea three years ago. Continued treatment until symptomatically cured. Admits he stopped treatment despite the fact his physician told him his prostate still contained pus. Two weeks previous to my seeing him he had attempted intercourse with a virgin who subsequently reported to him that she had dysuria and discharge. He had had no discharge for over two years and thought he was well. Smears from her discharge first failed to show any gonococci. His urine was clear and contained not over three to five pus cells per high power field. There was no urethral discharge. The prostatic fluid, however, was grossly semipurulent and microscopically loaded with pus, though diligent search revealed no gonococci. At first it seemed as though this was a case of transmission of a non-specific infection. A complement fixation test was taken on the man at a subsequent visit. The report came back "positive". Subsequently repeated smears were taken on both him and the young woman. Finally a definitely "positive" smear was found in her discharge, but the gonococcus was never observed in his smears. However, had it not been for the lead given by the complement fixation test, a positive diagnosis of gonorrhea might never have been made on either party.

CASE 2. W. B. History of gonorrhea fifteen years ago. Subsequently married; has two children; wife said to be well. Extramarital exposure occurred ten weeks before he came to the office. Had treated himself with injections and apparently pushed his infection posteriorly. Repeated urethral smears showed pus and mucus but no gonococci. Prostatic smears contained considerable pus. However, not satisfied that this represented a non-specific infection a blood for a complement fixation test was taken and it was reported "positive". I have not yet seen a "positive" report in a single case where there could be no question of the non-specific nature of the case. Therefore, I feel that here is another case where a diagnosis of gonorrhea depended on the use of a complement fixation test.

Wassermann reactions are always done on the same blood as the complement fixation test for gonorrhea. As Young states that syphilis may cause a "positive" reaction, it is important to be sure that the Wassermann is negative. Young also states "in most of the cases where absence of other evidence makes the complementary test most desirable it is difficult or impossible to confirm positively, at any later time, the results of the test; therefore, it should not be depended upon alone." But Barringer says "as far as my experience goes a complement fixation test either weak or strong is caused alone by gonococci inflammation." From this series, and past experience, I would agree with Barringer because, besides the "doubtful" and "positive" cases already mentioned, the gonococcus was found after diligent search on repeated smears in two of the other five male cases with "positive" bloods.

A "positive" blood does not necessarily mean that the patient is infectious but should arouse enough suspicion for one to be more than careful about clearing up all the clinical evidence of the disease. Case 1 described above proved to be infectious and a "positive" blood was the only evidence ever obtained on him. Naturally the two cases with "positive" bloods that subsequently showed gonococci in their smears were also infectious. On the other hand, it is generally accepted than an arthritis or other metastatic manifestation of gonorrheal origin will give a "positive" blood in the absence of any local evidence of the disease. Furthermore, McNeil and Jeck have clinical evidence to show that a "positive" reaction will persist for several weeks after a clinical cure. Therefore, if one has a patient that has been clinically cured, he should either wait a while before taking blood from him or expect to get an occasional weak "positive" that would be "negative" later without further treatment. It will thus be seen that there are many pitfalls in the proper interpretation or significance of even a "positive" blood.

Of the ten patients with "negative" bloods who had a past history of gonorrhea, three to ten years ago, but no evidence of it by smear in their present chronic urethral discharge or in their pus-laden prostatic secretion, none ever showed the gonococcus in subsequent smears. Therefore, it seems reasonable to interpret their present difficulty to secondary infection which had been superimposed on the old gonorrhea and which was now flaring up because of dietary or sexual irregularities or was metastatic from an acute infection elsewhere, usually in the upper respiratory tract. In this last group, as in the definitely non-specific group, the "negative" tests ran true to the clinical findings.

However, besides the false "negatives" obtained in fresh anterior cases and in certain cases of gonorrheal endocervicitis as mentioned above, there is another type of case in male patients where a false "negative" has brought the complement fixation test into bad repute. These are the cases where the patient is doing well, or is even apparently cured. He has had

no discharge for weeks and there is only a moderate amount of pus in his prostate. A complement fixation test if taken then may very likely be "negative". The patient is looking forward to an early dismissal when suddenly he notices a recurrence of discharge and a smear is positive for gonococci. These cases probably represent those who have hidden, silent pockets of infection in one or many of the urethral glands which may unexpectedly begin draining quite late in the course of the disease. Such cases, treacherously infectious, have "negative" bloods just as an early acute gonorrhea. It may therefore be appreciated that "negative" tests are so often totally unreliable that one should never wholly depend on them in saying that a given patient is free from gonorrhea.

SUMMARY: A series of thirty-seven private cases of deep chronic genital infection and seven cases of cured acute gonorrhea is reviewed on whom a complement fixation test for gonorrhea was used in the absence of any clinical demonstration of the gonococcus in an effort to possibly establish a positive diagnosis.

All cases that could properly be considered non-specific gave "negative" reactions. Some cases with a past history of gonorrhea gave a "negative", some a "doubtful", and some a "positive" reaction. In those that gave a "negative" reaction the gonococcus was never found. In all three of those that gave a "doubtful" reaction the gonococcus was subsequently found. In two of eight who had "positive" reactions the gonococcus was subsequently found; in another it was finally found in the patient's sexual partner, and in a fourth, who had salpingitis, the gonococcus was found in the husband but not in the wife. The reaction was negative at the time of discharge from treatment of all seven cases treated from onset of their acute gonorrhea on whom the test was used.

The value of the test in certain cases in establishing a positive diagnosis, the pitfalls in interpretation of a "positive" test, and the uselessness of a "negative" test alone, are discussed.

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DIATHERMY TREATMENT OF ACQUIRED BALANITIS, PHIMOSIS AND PARAPHIMOSIS

BY HAROLD NEIFELD, M.D.*

THOUGH diathermy has been employed recently in the treatment of gonorrheal urethritis, orchitis, epididymitis, vesiculitis and prostatitis, yet no reports of its use in balanitis, phimosis and paraphimosis have been recorded. In a series of twenty-five such cases, seen over a period of several years, that had received this form of therapy, the results have been so uniformly successful that the attention of the profession is directed to it.

The application is simple. The affected portion of the penis is placed in contact with and between two opposite metal electrodes, one applied to the dorsum of the penis, the other to its ventral aspect. The current is turned on, gradually increased to the point of greatest comfortable tolerance, and kept so for twenty minutes. One application often reduces the edema and swelling from one-third to one-half its pretreatment size, and always makes reduction of the prepuce possible. Three or four treatments are usually sufficient to cause complete disappearance of all enlargement. The electrodes must be carefully and accurately adjusted, the best contact in the above cases having been secured by scrotal electrodes held in firm contact with the edematous area by a clamp-holder.

That the procedure is sound is well supported by the clinical, pathological and histological evidence. According to Kovacs[1], the physiological and clinical effects of diathermy are best explained by heat production. Diathermy, by causing a local, deep-seated active hyperemia, promotes tissue drainage[2]. This hyperemia is due to a dilatation of the blood vessels, and to a diminution of the resistance of the blood in its passage through the arterioles and capillaries. Heat, by decreasing the viscosity of the blood, permits its passage through the vessels with less frictional resistance, so that the treated (heated) parts not only receive more blood per unit of time (volume), but the blood supply is also more rapidly renewed. The action of diathermy on infection is both direct (destruction or lowering of the vitality of bacteria) and indirect (increase of the power of the tissues to exterminate organisms)[3].

From a pathological standpoint, balanitis is an inflammation of the glans penis; when the edema and swelling become so great that the foreskin cannot be retracted, the condition is called phimosis. Paraphimosis, on the other

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hand, is that condition in which a long and tight foreskin is caught back behind the glans and cannot be retracted over the head of the penis[4]. Now, as these conditions are inflammatory, the signs of inflammation, dilatation of the capillaries with a larger though slower bloodstream, margination and diapedesis of the leucocytes, exudation of the serum and phagocytosis, are present[5].

Anatomically and histologically, the penis is admirably constructed for treatment with diathermy. The organ consists of three cylindrical masses of erectile tissue, the two corpora cavernosa, surrounded by a connective-tissue sheath (the tunica albuginea), lie side by side; and the corpus spongiosum (enlarged anteriorly to form the glans), surrounding the urethra, lies below and between them. The principal substances of the erectile tissue are the septa and trabeculae of connective tissue, the elastic fibres and the smooth muscle cells enclosing a system of communicating spaces (venous sinuses), lined by endothelial cells. The arteries of the corpora cavernosa with their well-developed musculature ramify through the erectile tissue and break up within the septa into a coarse-meshed plexus of capillaries. A few of these arteries empty directly into the cavernous spaces. Most arteries, on the other hand, form immediately beneath the tunica albuginea a rich capillary network that communicates with a deeper and denser venous network, which in turn gradually empties into the venous sinuses. There are also anastomoses between the arterial and venous capillaries, which communicate later with the venous network just mentioned. In the corpus spongiosum the arteries do not empty directly into the cavernous spaces, but first break up into capillaries. In the submucosa of the urethra there is a rich plexus (venous), while in the glans the arteries end in capillaries which empty into veins with well-defined musculature. The blood is collected by the venae emissariae, which empty into the vena dorsalis penis and into the venae profundae[6].

In conclusion, diathermy by inducing an active hyperemia with its consequent increased tissue drainage reduces edema and swelling in and produces cure of balanitis, phimosis and paraphimosis.

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MASSACHUSETTS TUBERCULOSIS LEAGUE

HEALTH EDUCATION DEMONSTRATIONS IN MASSACHUSETTS HIGH SCHOOLS*

BY FRANK KIERNAN, A.B.†

SINCE the epoch-making Joint Report of the American Medical Association and the National Education Association in 1924, which declared the health of the pupils to be the first responsibility of all schools, and since the United States Bureau of Education's statement of 1918 —that health needs cannot be neglected during the period of secondary education without serious danger to the individual and the race, the necessity of health education in the high schools of America has remained a constant challenge.

We are not proud of our record in health education in the secondary schools in Massachusetts in these intervening years. Although spasmodic efforts were made in this field by our organization, it was not until 1929 when the National Tuberculosis Association offered to the Massachusetts Tuberculosis League a thousand dollars for a high school health education experiment, that we undertook definitely to work on this problem. We were further led to action by the publication of statistics by the National Tuberculosis Association bringing forcibly to the attention of the country the high mortality from tuberculosis in the adolescent years and in early maturity, especially among young women. Realizing that the promotion of health education in the secondary schools might be a means of stimulating efforts to control this high death-rate, the League saw the opportunity presented, and proceeded to undertake the inauguration of a practical experiment in the teaching of health facts and the practices of hygienic living to high school students.

Due to especially favorable local conditions, meaning thereby keen interest on the part of the superintendent and assistant superintendent of schools, and the principal of the English Senior High School, the experiment was started in Lynn. This has gone on for three years. After the first two years, during which time much was learned by trial and error, under similarly fav-

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orable auspices an experiment was inaugurated in the Richmond Junior High School of Danvers.

In the schools of both cities there have been developed procedures including the following: A—Health Protection, including physical examinations, x-raying and follow-up of suspicious cases, social histories, home visits; B—Health Promotion by means of faculty and student health councils, with appropriate committees on personnel, program adjustment, and sanitary care of the school building; C—General Health Emphasis Teaching by integration of teaching with school cafeteria, assemblies, radio talks, debates, essays, posture contests, campaign for the correction of dental defects, school newspaper and school library; D—Health Instruction by Means of Experimental Courses in Classroom. Here were developed units in physiology underlying personal hygiene, physiology and nutrition in its relation to health, and community hygiene and public health. The material for these units has been based on high school pupils' interests, facts not learned in elementary schools, as revealed by objective health knowledge tests, habits needing to be improved as shown by health habit surveys, morbidity and mortality tables, and the opinions of health education experts. One of the teaching units worthy of special mention is that on tuberculosis, its cause, treatment and prevention.

We felt in the development of our experimental health teaching that we could not trust to correlation of subject matter, for correlation in and of itself is likely to be too casual. Therefore, we have developed teaching units which involve thoughtfully organized, planned teaching, at the same time guided not only by what the teachers have in mind the pupils need to know, but based first of all on what the pupils are interested in finding out about health. Here our work has progressed along lines similar to the course of study in health education for the secondary schools developed by the Ohio State Department of Education, the project which, as you probably know, was sponsored and partly financed by the Ohio Public Health Association. From the information we have obtained from an objective health knowledge test given to students entering high schools, we are convinced of the fact that there is much factual material in health which must be taught the older child in the secondary schools. The importance of this has recently been called to our attention by Dr. H. E. Kleinschmidt of our National Association, who pointed out that health habit training and health publicity, important as they are, are not sufficient to insure an enlightened, well-informed public opinion in matters pertaining to health. Dr. Kleinschmidt aptly says: "If the particular health idea we wish to introduce to the public is a bit complex, health publicity helps but little. For example, in the present state of our knowledge of tuberculosis, it seems very important that parents should understand the difference between the adult type and the childhood type of tuberculosis, but how can we teach them that distinction unless they possess a working knowledge of physiology and hygiene?"

Three years have gone by since this work was begun and while the experiments are, of course, not yet completed, we are able to see certain definite results and to confirm our belief that health education will remain a vital part of the programs in these high schools. Not only do the school administrators and teachers believe in the procedures developed, but the parents, as well as the students themselves, are enthusiastic. We feel that we have developed methods and materials which may be used for guidance in other places. This material is now being tried out in other high schools, and we are anticipating the publication of a report. Already under the direction of the League, several other schools in the state have begun to develop experimental health education programs.

To secure additional data as to the age levels in which the various health topics might best be presented, experimental teaching is now going on in its ninth year at the Brookline High School. Next fall we plan to continue this and other experiments to determine, if possible, where it is best to introduce these topics. It may be that this task is one for a detailed research study.

Because of the leadership of our Plymouth County Health Association, a study is now under way in the town of Rockland. Also, the Massachusetts League has begun a coöperative plan with the Southern Worcester County Health Association for the x-raying of students in the smaller towns of the county, in which project the educational aspect will receive important emphasis; this because we realize that testing in and of itself is of little value unless health education precedes and follows. May I stress this point here, that physical examinations may be made more than physical examinations. They may be made educational by developing in the pupil-examinee a new and more intelligent understanding of the body and its functions.

As a means of keeping the undertaking on a sound pedagogical and medical basis, an Advisory Committee on Health Education Problems in the Secondary Schools was formed more than three years ago. It is composed of school authorities, members of the State Departments of Public Health and Education, and representatives of Schools of Public Health of Harvard and the Massachusetts Institute of Technology, and the Simmons and Boston University Schools of Education. This advisory group has proved a useful medium for an exchange of thought not

only as to "the why" of health education, but as to "the how" of carrying out the programs in the schools. In this undertaking the League could not have accomplished significant results without the initial support of the State Departments of Public Health and Education. This has made it possible for the private and official agencies to work hand in hand in bringing the matter before the public. However, because of the fact that the Tuberculosis League has a full-time trained Educational Secretary, it has been the agency which assumed the leadership and supervised the carrying on of the work. The staff of the Health Education Department of the League has remained the same, with the exception of the health counsellor placed in the Lynn High School, whose salary for this school year is being paid partly by the League, and in part from the grant of the National Tuberculosis Association.

It was realized from the start that any undertaking in the schools should be worked out in a way which would be practical enough for the school authorities to administer, and to continue after the private agency had withdrawn. For this reason, the demonstrations which we have been conducting have involved little additional expense. Even before the depression came, they were not planned to require an elaborate set-up, but were attuned to the present teaching personnel, and made to articulate with the existing school program.

In Lynn and Danvers intensive in-service training has been carried on by the League for the regular teachers who are assigned to health classes, and who will continue to direct the health instruction and act as members of the health councils.

Also, for the past three years as an important supplementary activity, special courses in health education have been established and underwritten in the local colleges and universities for which, out of our regular health education budget, the League has awarded tuition scholarships. School superintendents, high school principals, teachers of the sciences, physical education and home economics, teachers of continuation schools and nurses have attended these courses.

A further step of the League's advisory group has been that of the survey it conducted to ascertain the present status of health teaching in secondary schools, the findings of which have led to the formation of a committee of experts called into being by the State Commissioner of Education. This new committee plans to develop an official state course of health education for high schools. It is made up in part of members of our own Advisory Committee, the Educational Secretary of the League continuing to act as active secretary of both committees. This last feature we consider one of the most significant results of this whole undertaking.

We are hoping to be invited to give a more complete report of this entire program of teaching health in the secondary schools at another annual meeting of the National Tuberculosis Association.

Two or three times in this paper, you have heard mention of money expended in connection with this work. We want to stress very forcibly that indispensable as were the grants from the National Tuberculosis Association in giving us an impetus for these experiments in Massachusetts, we are convinced that given the vital enthusiastic interest in health education on the part of the tuberculosis associations, and a wide-awake interest in health on the part of the school administrators, health education can be introduced in a very definite way, and made part of the fabric of the school curriculum without any considerable expense.

At the annual meeting of the League in April of this year, a special program was given, at which the assistant superintendent of schools of Lynn was one of the principal speakers and the superintendent of the Danvers schools the other. Both paid high tribute to the work which has been carried on by our League, as described in this paper. Interesting as were the addresses of these gentlemen, they were not more interesting than those of the Lynn high school senior and the Danvers junior high school student, who testified in characteristic high school language as to their appreciation of what health teaching meant to them. The girl said in part, "I will be more careful to practise good health habits now that I understand some of 'the why's' behind them." The boy said, "I chose this course in health even though it was an elective, because I wanted to have at least one thing from my high school course that would be useful to me after graduation."

THE NEW HAMPSHIRE MEDICAL SOCIETY

THE CONDUCT OF NORMAL LABOR*

BY BENJAMIN P. BURPEE, M.D.,† MANCHESTER

WHEN we are first called to see a case at the beginning of labor, there are two questions that enter our minds, either consciously or subconsciously; first, how hard a case is it going to be and secondly, what can we do to relieve, as much as possible, the pains of childbirth. The first question, however, should not be necessary. We ought to have a pretty good idea, before the time for labor arrives, as to what sort of a labor it is going to be. If the patient is a primipara, a previous examination a week or two before the expected date is essential. From this is determined the position of the fetus. It is extremely necessary to know this, because the treatment of a given case depends entirely upon the position of the fetus. It is very generally agreed that a large percentage of the difficult cases, those in which failure of the head to descend or to flex properly, and those in which difficult forceps deliveries occur at the end of the second stage, with damage either to the fetus or to the mother or with actual failure to deliver at all with forceps, are due to unrecognized posterior positions, which, had they been diagnosed earlier, would have received entirely different treatment. Thirdly, a rough idea should be gained as to the size of the pelvis, chiefly as to the inlet and the outlet. Personally, I do not put too much stress upon the pelvic measurements. In the first place, I have never felt that it was an easy thing to measure a pelvis. The personal equation enters very largely, and I think that the measurements by different men will show a considerable variation. Also it is an assured fact that the best pelvimeter is the fetal head, so that I have always felt that an estimation as to the size of the pelvis is better made shortly before labor than it is during the first few months, inasmuch as at the time of labor the relation between the size of the pelvis and that of the fetus can be more accurately estimated; also, it is not unusual to find a pelvis that is quite a bit justo-minor where a difficult labor might well be expected and yet have the delivery surprisingly easy. If, at this time, a week or two before the expected date of confinement, the head is found low in the pelvis, or at least a low mid on rectal examination, we certainly have nothing to fear as regards disproportion between the size of the fetal head and the size of the inlet. I find that I am often able to gain a considerable amount of information in making this rectal examination, if there is any question of the head not being well engaged, by asking the patient to bear down. If the head is felt to descend slightly with this contraction of the abdominal muscles it is pretty good proof that the pelvis is of satisfactory size. If the fetal head is not well engaged a week or so before and most certainly if it is not well engaged at the onset of labor pains, I feel that it is an ominous sign. I was interested recently in reading an article by a French author in which he stated that in only five per cent of primiparae did engagement occur at the beginning of the ninth month, seven per cent during the thirty-ninth week, thirty-four per cent during the last week and over fifty per cent not until labor had actually started. I most certainly would not agree to that; at least it has not been my experience. I can remember offhand of only one case in the last dozen years in which a non-engaged head in a primipara at the onset of labor ended with anything that approached the semblance of a normal delivery. On the other hand I can remember many that, following the usual test of labor, were definitely committed to vaginal delivery, where many times I wished that section had been chosen. I believe that any slightly engaged or floating head at the onset of labor in a primipara is a loud cry for section, or at least a test of labor in which the membranes are preserved insofar as possible and vaginal examinations are rigidly avoided with the possibility and probability of section in mind.

If no disproportion is found between the head and the inlet, we are concerned with the outlet. This may be fairly well estimated by determining the distance between the two tuberosities and by a rough estimation of the pubic arch in order to rule out a funnel pelvis which may be just as troublesome in the termination of a labor as a disproportion at the inlet.

Another factor that must be taken into consideration is the age of the patient. It has been shown that fifty per cent of primiparae over forty years of age have a labor of over twenty-four hours and that one third of them end in forceps deliveries as compared with seven per cent of younger patients delivered by the same men. DeLee advances a very interesting observation with regard to this, namely, that elderly primiparae conceiving at once after marriage are apt to have an easier labor than those that have been married a long time before conceiving.

Therefore, in spite of the fact that it is quite difficult to estimate the time element exactly in obstetrics, we should have a rough idea before

*Read at the annual meeting of the New Hampshire Medical Society at Manchester, May 17, 1932.

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the labor begins as to what to expect and should most certainly know in what position the fetus is lying.

All of us who do obstetrics undoubtedly try to lessen or relieve the pain of the woman in labor. In the first place this is only humane and in the second place, women have in the last few years come to expect definite mitigation of suffering, and perhaps, unfortunately, are apt to gauge their obstetrician not so much by the end results or by the obstetrical skill as by the amount of pain experienced, so that it becomes an economic necessity for the man doing much obstetrics to relieve pain to as great a degree as possible and to make the labor short, this of course within certain rational limits. I was interested recently in visiting some of the large lying-in hospitals, in noting the number of gas machines and the large supply of gas cylinders about the delivery rooms, where a dozen or fifteen years ago there was not a single machine, and in fact any anesthetic was forbidden without a member of the staff in attendance. I was told that even straight charity patients were now given as much gas as they required. Any analgesic or anesthetic agent used during labor should, I think, answer three requirements: Is it safe for the fetus, is it safe for the mother and does it retard labor?

When should analgesics be begun in primiparous labors? The patient is probably better off at home until the pains are coming every six or eight minutes, and it is my custom to try to dissuade them from going into the hospital before this time. Nothing can be done for them if they go in too early and they are very apt to become discouraged if they are forced to stay at the hospital for a long time before active labor begins. By the time that the contractions are coming every four or five minutes they are usually becoming painful and analgesics should be started. My personal preference is for morphine and hyoscin. After what may perhaps be a rather too brief experience with other methods to offer an intelligent criticism, I feel that this is the best combination. Without question this should not be given if delivery is to be expected within three hours, because of the danger of asphyxia in the baby. However, as sometimes happens, delivery may occur more quickly than is expected without any sign of asphyxia in the fetus. If a mild degree of asphyxia is encountered, I do not feel that this need usually cause a great degree of alarm. I have used a great deal of morphine-hyoscin and I do not believe that I have ever lost a baby from this treatment. I am, however, very strongly opposed to the use of morphia just before section, where so short a time elapses between its administration and the birth of the child and have seen at least one case where I felt certain that the death of the child was directly and solely due to the use of this drug. The mild degree of asphyxia that may result occasionally in vaginal deliveries, and this applies to any drug, responds quickly to the proper gentle methods of resuscitation and the alarm that it inspires is in no way comparable to that felt when the asphyxia is due to venous congestions or to trauma. The criticism has been advanced against morphia that it lessens the frequency of the contractions. This may be true, but the pains pass off more slowly so that the actual amount of work done by the uterus is as great or even greater.

The beauty of hyoscin, of course, lies in the amnesia that it produces. It has no ill effect on the baby and apparently tends to shorten labor, perhaps through its relaxing effect on the cervix. The usual dosage of these drugs is gr. 1/4 of morphia and 1/150 of hyoscin. As soon as the hypodermic is given the patient should be put in as quiet a place as possible. The room should be darkened and it is also well to put cotton in the patient's ears. It is quite useless to give these drugs and then put the patient in a noisy open ward. Usually the patient is good for from three to five hours of fairly comfortable labor following this. Not infrequently complete dilatation occurs and the head is found to be on the perineum before she again begins to complain very much about the pains. Rarely it may be found that considerable restlessness occurs following the morphia but usually the patient is pretty groggy. She may rouse a little at the height of a pain and then sleep between that and the next one. I almost never repeat the morphia except in an unusually protracted case but do occasionally repeat the hyoscin.

Even if the patient is not ready for delivery by the time that the morphin-hyoscin has worn off she has practically always made a very satisfactory advancement and if I find that there is some dilatation still to be accomplished, I usually fall back on sodium amytal. Next to morphin-hyoscin I have had the best results with this and it has the advantage that it may be given without regard to how soon the baby is to be born. It can be given in fairly large doses and it has been shown that it works as well when given by mouth as when given intravenously. Hamblin states that less than nine grains should not be used as the initial dose and that it is safe to give as high as thirty grains during a single labor. I was rather amazed to be told at two of the largest lying-in hospitals in New England that they held sodium amytal in considerable fear due to the mania that it produced. At one of these they stated that they did not use the drug without a special nurse in attendance. Personally I have given it in perhaps two hundred cases and in only one have I noted mania that was annoying. This occurred recently in a rather protracted primiparous labor. Mania also resulted

from morphin-hyoscin and from hyoscin alone in the same patient so that I should attribute it to an idiosyncrasy in the patient rather than to that particular drug. It does not protract the labor and it does not tend to produce hemorrhage. I have never seen it have any ill effects on the baby although I doubt whether I have ever used more than eighteen or twenty grains in any one case. My initial dose is usually six, sometimes nine grains. At times there may be a slight inclination on the part of the baby to be a bit dopey at birth but this always clears up quickly.

Avertin in my experience has been rather disappointing. In the first place, I found it a nuisance in that it prevented my following the degree of cervical dilatation by rectal examination. Also it frequently leaks out of the rectum. It produces a very satisfactory analgesia, but to my mind it is too short. It rarely lasts over an hour. Theoretically this should be the last hour of labor, but I found it difficult to guess when the last hour of labor was. Perhaps the fact that I am not keen for a long second stage was one reason why I did not care for it. I think the analgesia is better than that from sodium amytal but I do not think that it is as safe. I must admit that I cannot view without a considerable amount of concern a systolic blood pressure that drops into the sixties even though this may be controlled with adrenalin. Avertin seems to work much better in surgical than in obstetrical cases. After watching a few patients prepared with this drug for surgical operation I had expected a great deal from it in obstetrical use. However I think that this may be explained by the fact that a surgical operation is done at a previously determined moment which obstetrical deliveries cannot be; also that the previous preparation for surgery is better, the dosage larger and the effect of the drug may be accentuated by the use of morphia.

J. P. Cohen advocates the use of hyoscin with sodium amytal, omitting the morphia. He gives the two drugs together in the initial dosage, repeating the hyoscin once if necessary in two or three hours and the sodium amytal every two or three hours as necessary. With this particular combination I have had no experience, nor have I had any experience with the synergistic method of Gwathmey, combining rectal oil and ether with morphia and magnesium sulphate. I confess that I do not care for ether as an anesthetic during labor except where actual delivery is contemplated because of the fact that it tends to stop the pains. The same criticism may be offered to the use of chloroform in addition to the fact that it is the least safe of any of the anesthetics and has the most injurious effect on the liver, an organ already apt to be damaged during pregnancy.

As the end of labor approaches and the effects of the analgesic have begun to wear away, it may be desirable to give the patient an anesthetic. Nitrous oxide-oxygen is without question the anesthetic of choice. It is not only the safest but it has the added advantage that it does not slow up the pains, in fact it very often makes them stronger. It is not expensive. I would say that a patient rarely consumes more than five dollars worth of gas per hour. It has no effect whatsoever upon the fetus, because of the fleeting effects of the gas and its combination with oxygen. In fact the gas machine is useful after delivery and before the placenta has been detached in that it is possible to flood the fetus with oxygen by feeding it to the mother combined with eight to ten per cent carbon dioxide to stimulate respiration. It is possible to give gas-oxygen for any number of hours without ill effects to the mother or fetus. I do not care for gas-oxygen as an interrupted analgesia, giving the gas at the beginning of a pain and stopping when the pain is over. My objection to this is that very many times the patient gets no relief until the pain has gone, then lapses into a short unconsciousness and, when she comes to, is disappointed because the baby has not arrived, not realizing how short has been the quiescent period and this happening repeatedly thereby causes discouragement to the mother. This is also one of the objections to the use of ether, which cannot be given continuously to the point of anesthesia without stopping the pains. I think that it is much more satisfactory after the gas is once started to keep the patient under it. It is not a difficult thing to give gas-oxygen satisfactorily for obstetrics. Any nurse with a little experience can do it well.

If the patient is a multipara she is just as much entitled to and just as appreciative of an analgesic during labor as the primipara. However, the comparative shortness of multiparous labors with those of primiparae, necessitates care being taken in the selection of an analgesic and when it is given. Many multiparae are sufficiently dilated when first seen so that gas may be started. If there is very little dilatation morphine-hyoscin may be used if the pains are strong and the time likely to elapse seems more than three hours, otherwise sodium amytal as with a primipara.

What about examinations during labor? That brings up the old question of rectal versus vaginal examinations during labor. I must confess that I could never understand why properly conducted vaginal examinations should increase the likelihood of infection. If there are pathogenic bacteria in the vagina at the time of labor they will eventually make their way into the uterus regardless of whether they are pushed ahead by the examining finger. It is assumed that a vaginal examination will be carefully made using sterile gloves and touching nothing external to the labia minora. If pathogenic

bacteria are not in the vagina they will not or should not be introduced by a properly conducted examination. Some interesting figures have been presented from the New York Lying-in where it was shown that in a series of some 80,000 cases, there was no increased morbidity in spite of frequent vaginal examinations. This is a teaching service and undoubtedly the examinations were by students under supervision. It was also shown that a decreased morbidity did not result from limiting vaginal examinations. Personally I prefer rectal examinations to vaginal—perhaps because they are easier and because one's conscience is clear if anything does happen. It is not difficult to determine anything that you want to know in the vast majority of cases. Occasionally when the cervix has been thinned out to a very great degree and especially if this is combined with a very small amount of forewaters, it may be difficult to determine the amount of dilatation. Also there is more difficulty in determining the sutures and fontanels and danger in overlooking what has been called a concealed second stage. By this is meant a cervix, which, although potentially completely dilated is still easily felt by rectum. The findings that should lead one to suspect such a condition are a peculiarly soft cervix that hangs down from the presenting part like the open end of a bag, which may be easily moved from side to side and which disappears during a pain only to reappear again. Also if it is desired to rupture the membranes, it is of course necessary to resort to vaginal examination.

Not so very many years ago our teaching was that it was a most heinous crime to rupture the membranes before complete dilatation had occurred. This, to my mind, is most erroneous. There is a large class of multiparae in whom dilatation of the internal os with complete taking up of the cervix has occurred before actual labor starts and yet the first stage of labor will drag, the pains appear useless, irregular and slow. In such cases I feel that the woman may be saved much unnecessary suffering by rupturing the membranes at once and thus by allowing the head to drop, start active, useful labor pains. I must confess that, however unorthodox it may seem, it is my custom to rupture all multiparous membranes at least by the time that half dilatation has occurred, unless some definite contraindication exists. For example, a breech presentation where the possibility of prolapsed cord would make it advisable to preserve the membranes until actual delivery was possible, a posterior position where the preservation of the membranes is useful in obtaining proper flexion and rotation and all cases where a long thick cervix and an undilated internal os is a definite contraindication.

We must carefully watch during the course of a long hard labor the inroads made upon the reserve strength of the baby and mother. It is essential to keep up the mother's fluid and carbohydrate intake in order to prevent what, for lack of a better term, we may call an acidosis.

The condition of the fetus may be watched through the fetal heart and this should be listened to at frequent intervals. Although it is probably true that hard and fast rules cannot be applied to a case of labor, still we must have something to go by. Tweedy's rules of the test of labor consist in taking the mother's pulse and temperature every two hours and counting the fetal heart every two hours. When the temperature rises above 100°, interference is indicated in behalf of the mother and, if the fetal heart rises above 160 or drops below 120 on three consecutive counts at minute intervals, interference is indicated on behalf of the baby because of the danger of intracranial hemorrhage. It is claimed, that in 14 years in which this test of labor has been used at St. Mary's Hospital in Philadelphia, these rules have failed to warn in only 13 per cent of 231 cases that eventually ended in serious complications of labor.

I believe that pituitrin has a very definite place in the second stage of labor. There are many flabby uteri where it does no harm to stimulate the force of the contractions with this drug. There are certain indications that must be met. It must not be given where there is any obstruction either from the bony pelvis or from rigidity of the maternal soft parts. Hence it should not be used during the first stage except at the very end in a multiparous labor where the remains of the cervix are extremely elastic and the position is anterior. It should not be used in primiparae at all because of the rigidity of the maternal soft parts. It should not be used if the position is posterjor because of the possibility that rotation might not occur without operative interference. It should not be used at the end of a long hard labor before the third stage, nor when there is any question of the lower segment of the uterus being thinned out. The dosage should be rational. Once given it cannot be recalled. Three or, at the most, five minims is sufficient. Yet in many multiparae during the second stage, it is a safe and useful drug, not only for its expulsive effect but also as an aid to the prevention of after-pains.

I am in favor of shortening the second stage as much as is consistent with safety. By this I mean the routine use of control or prophylactic forceps and of the episiotomy in primiparae. I believe that it is much safer, once the head has reached the perineum and internal rotation has occurred to make a space for it to pass through by an episiotomy. This obviates the necessity of dilating the perineum at the expense of the fetal head and decreases the pos-

sibility of intracranial hemorrhage. I believe that there is less danger from a simple low forceps with a proper parietal application than there is from an hour or two more of labor. Moreover there is a decreased amount of wear and tear on the mother and a preservation of the tonus of the perineal tissues. It is not a difficult thing to deliver a fairly large per cent of primiparae without lacerations. Yet I think that we have all seen cases later which have not been torn at labor and still present large cystoceles or rectoceles. I believe that these are prevented by the routine use of the episiotomy since the tissues are not stretched to the point of losing permanently their tonicity. The incision is a clean cut one and repair leaves a strong firm perineum. Episiotomies may be either median, mediolateral or lateral. Personally if the head is on the perineum and completely or almost completely rotated, I prefer the median incision because I feel that it heals better and gives a more satisfactory result. If the head is high in mid-pelvis and the delivery a fairly difficult one, I prefer the lateral incision since it gives more room. If the perineum is a small one, say less than an inch and a half high I would warn against the median incision, since it is apt to result in a third degree tear if there is the slightest extension of the episiotomy. In such a situation with a small perineum, I should prefer a mediolateral incision. In repairing the median or mediolateral incisions for the past two or three years I have used a method that may perhaps sound a bit strange but which has given end results that have been extremely satisfactory, more so than those obtained by sewing up in separate layers. I use only silkworm gut in the nature of stay sutures without any catgut inside. I put these in before the episiotomy is performed, either three or four in number, depending upon the length of the incision, starting at the lower end of the incision and going through the entire perineal body into the vagina, across, and out at a corresponding point of the opposite side. The top suture should be about a quarter of an inch above the fourchette. The result is that when the tissues are pulled together again everything is in exactly the proper place. After the stitches are put in the fingers may be inserted into the vagina to protect the silkworm from being cut when the episiotomy is made. Then it is necessary to put snaps on either end of each suture in order to make sufficient room for the birth of the head. The stitches should not be tied until the placenta has been delivered. The results as I have said have been very good.

When forceps are applied and traction is made, I think that it is better to pull the head down to a point where it has completely slipped down beneath the symphysis, then remove the forceps and deliver the head manually. Care should be taken to protect the perineum just the same as with a normal delivery in order to prevent a tear from increasing the incision. After the head has been delivered no traction should be made on it, because of the danger of damage to the brachial plexus. Having had three birth paralyses within a short time several years ago, I learned that it is better to wait until either the nurse has pushed down on the fundus and brought the anterior shoulder out from beneath the symphysis or until a contraction has done this. In either case there is no appreciable delay.

DeLee has called attention to the fact that there are more deaths from accidents in the third stage than in the other two combined. Therefore the technique of the third stage should not be overlooked. It should not be hurried. Great care should be taken to have the fundus held properly. It is possible to have quite an extensive hemorrhage within the uterus if the nurse does not understand exactly what to do. Personally I prefer to use pituitrin either at the end of the second stage or immediately following the birth of the child, in order to prevent excessive loss of blood. It is an undoubted fact that the less blood lost during the third stage, the more satisfactory is the puerperium. It has been shown that except for the size of the baby and the stature of the mother, which have very slight effect on the blood loss of the third stage, the only factor that does influence the blood loss is the length of time that elapses between the separation of the placenta and its expulsion. The placenta should not be expelled too early by too vigorous use of the Credé method because of the likelihood of hemorrhage. Neither should it be allowed to remain within the lower part of the genital tract after it has separated. Traction on the cord should never be made because of the increased risk of inversion. Too vigorous massage of the uterus is not necessary during the third stage, in fact it is better to massage only the anterior surface of the uterus below the fundus. This promotes contractions just as well and does not tend to push the uterus downward, in fact quite the opposite. When light Credé is used to expel the placenta, it should consist chiefly of an anteroposterior squeezing with very little downward pressure. After the placenta has been expelled, I think that it is a very good idea in multiparae to have the nurse hold the fundus for an hour. This tends to give the uterus a chance to regain its tonus and to prevent it from filling up with large clots, thus both preventing hemorrhage and being particularly useful in the prevention of afterpains.

Discussion

Dr. Donald G. McIvor, Concord: I think we all ought to be very grateful to Dr. Burpee

for this paper. He certainly gives us some very practical statements about labor. I think one thing he has brought out that is very important is the preliminary examination a week before. We thus know just what we have to deal with when labor does start. Today it is quite true that we have to meet the wishes of the public in trying to relieve the patients from labor pains as much as possible. Personally, I have used very little sodium amytal. I have used some of the opium derivatives, particularly pantopon, with primiparae in the early stages. I have never used it with multiparae. The drug I have used with a great deal of satisfaction has been nembutal, which is similar to sodium amytal but I think it is more potent than sodium amytal. This, given to multiparae, followed with hyoscin, has given very satisfactory analgesia, combined of course with gas. I do not think any of the drugs can be used alone.

Concerning examinations I have always employed the vaginal route and have had no trouble. I occasionally do a rectal examination but I have never been able to obtain the information I wanted by that technic.

I have never seen any trouble with pituitrin in small doses, and have even given it to primiparae at the end of the second stage, in small doses.

I have used episiotomy with primiparae as a matter of routine and agree with Dr. Burpee that it does save rectoceles because you have a clean cut and a strong perineum afterwards. I have never used his method of putting sutures in before delivery of the child. I think that episiotomy done when the head is on the perineum, followed by pushing on the fundus by the assistant during the pain, has very often saved the use of forceps, and it certainly lessens the time of labor.

Pituitrin, in postpartum hemorrhages, I have used with saline intravenously, and should like to ask Dr. Burpee if he has had any experience giving it in this way. It certainly stops the hemorrhage and saves packing.

Dr. Chester F. McGill, Portsmouth: Dr. Burpee began his very comprehensive and timely paper with a discussion of the occipitoposterior position. This I think still continues to be the most frequent and probably the most troublesome complication of labor. He made a plea for a better and earlier diagnosis. There is no doubt that the whole story of the troubles that do later occur with a delivery of this kind are due to failure to make the diagnosis earlier. The cases are delayed and long protracted, and are very important factors which suggest the probability of the posterior position. There is no doubt at all that facility can be gained with a little practice in palpation and discovering the position.

The feeling of the anterior shoulder away from the median line and the sounds of the fetal heart in the mother's groin are probably the outstanding factors in connection with the diagnosis. It may be possible and ought to be tried by everybody, I think, to convert it and change it into an anterior position. Of course, it will not always be successful, in fact it probably will succeed in only a few of the instances, but the method of Watson, putting a folded towel with a binder between the anterior spine and shoulder, is advantageous. That is simply to be mentioned and little is to be expected from it.

Since the examination below meets certain difficulties in differentiation of the various sutures and fontanelles, it probably is true that, since the results are no better from the point of view of infection when the vaginal examination is avoided, we will continue to do vaginal examinations. It is difficult enough then.

Williams suggests, upon being unable to determine accurately the position, that the posterior ear be located. What a sensible thing for the average man, particularly one who is casually doing obstetrics! He also suggests that, following the application of forceps, they be taken off and the hand inserted and the posterior located.

As to analgesia, I think as a matter of practical obstetrics that sodium amytal is the best, chiefly because the cases will be observed at the time when it will be absolutely improper to use morphine as a practical matter.

Dr. Burpee mentioned avertin and his disappointment in its use. I recently heard a very competent obstetrician of Worcester, Mass. of some 16 years' experience, say that he counseled against its use in obstetrics. He was very keen about it in gynecological operations, however, and reported its use in some 750 cases.

Dr. Richard W. Robinson, Laconia: I know we are all creatures of prejudice and for that reason I want to say a word because I do absolutely believe that a stronger brief for the practice of rectal examination can be made than has been made manifest by Dr. Burpee in his paper.

In the first place I think it has been very definitely and conclusively proved years ago that the organisms that ordinarily cause dangerous infection following childbirth occur frequently in the normal vagina but only in approximately its lower third. These bacteria, principally the streptococcus, the staphylococcus and the colon group have not wings and cannot fly. They produce their infection only upon areas that have been traumatized and the infection progresses through the blood and lymph channels. Following labor, it seems entirely reasonable that the normal drainage of the uterus and vagina being downward and outward, the organisms that lurk harbored in the lower vaginal tract are practically incapable of harm providing

they have not been carried upward by such agents as the fingers and forceps.

If in the matter of determining accurately the position of the fetus, vaginal examination was the only procedure it would be entirely different. Perhaps the danger of infection is not so great as has been generally thought. Yet certainly, in most cases the combined use of external palpation and rectal examination determines the position just as well as examination by the vaginal route. It does not require the careful preparation of the hands or of the perineum that vaginal examination makes necessary. So that for these reasons I think it is the procedure of choice in the majority of cases. It is the safer procedure for the patient and the easier procedure for the physician.

Dr. Ezra A. Jones, Manchester: I was not present in time to hear this whole paper but I did hear Dr. Burpee speak about obstetrical paralysis, which I believe is an important subject. The doctor, I believe, stated definitely how this paralysis is caused. I remember very well the first two cases that he had in his practice here and we discussed the exact method by which this paralysis is produced. Most men have contended that it is due to stretching of the nerve trunks by pulling the head away from the shoulder and I think Dr. Burpee has proved definitely that this is true. During manual delivery of the head if undue traction is made away from the anterior shoulder, which is caught under the symphysis, injury to the cervical nerve roots may follow. The fact that Dr. Burpee has had no more cases since he demonstrated this, I think proves that this theory is correct and I want to congratulate him.

Dr. Herbert S. Hutchinson, Milford: *Mr. President*—I should like to ask Dr. Burpee if he has ever had any experience with a preparation known as thymophysin, which is to be used instead of pituitrin, the claim of the manufacturer being that it can be repeatedly used at short intervals in a case of labor where the pains are inefficient and slow, and that by means of this preparation you can get a result which approximates normal labor? You get good results in the end, and there is no danger such as may happen through a careless use of pituitrin. I have had occasion to use thymophysin in two or three cases with very gratifying results, but I do not know whether I like it better than pituitrin in the long run. I would like to ask the doctor if he has had any experience with this drug.

Dr. Burpee: I have had no experience at all with thymophysin. This is a combination of thymus and posterior lobe of the hypophysis. According to the literature the consensus is that it has no advantage over pituitrin, and it has every disadvantage that pituitrin has. It cannot be used where pituitrin is contraindicated.

With regard to the use of pituitrin intravenously in postpartum hemorrhage, I have not used it for that purpose. I think I may have been lucky with postpartum hemorrhage (I have not had many cases), but I think that is due to the fact that I use pituitrin as a routine in every case, either at the end of the second stage or at the beginning of the third stage.

Author's note: Since the delivery of this paper I have had considerable experience with the above mentioned method of Cohen discussed by him a year ago in the symposium on analgesics and recently reported from the Boston Lying-In Hospital. I am extremely enthusiastic over the results obtained.

B. P. B.

ANOREXIA NERVOSA*

BY FRED ELLSWORTH CLOW, M.D.† WOLFEBORO

THE most striking disturbance of the appetite and digestion, occurring probably as a psychoneurosis is anorexia nervosa, first described sixty years ago by Sir William Gull. Lasegue, in 1874, independently studied the same disease, but ascribed its cause to tuberculosis. Gull, as well as Osler, considered the disease a manifestation of hysteria. Within recent years investigations have been made to establish a metabolic or endocrine etiology, but there is still considerable divergence of opinion. All are agreed, however, that anorexia nervosa is a definite clinical entity, frequently of serious character; always slow in its development; always difficult of treatment; and not rarely fatal.

*Read at the annual meeting of the New Hampshire Medical Society at Manchester, May 17, 1932.

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Disorders of the appetite have long been known to occur in certain types of people particularly those of unstable nerve mechanism and function. Secondary disturbances of serious type occur in the insane. But, in anorexia nervosa, quoting Weir Mitchell, "to call it loss of appetite but feebly characterizes the symptom. It is rather an annihilation of appetite, so complete that it seems in some cases impossible ever to eat again."

Jelliffe, in a translation from Dejerine (1907), gives the classic picture which can but arrest the attention of the most casual student:

"It sometimes happens that a physician has patients,—they are more apt to be women—whose appearance is truly shocking. Their eyes are brilliant. Their cheeks are hollow, and their cheek bones seem to protrude through the skin. Their withered breasts hang from the

walls of the chest. Every rib stands out. Their shoulder blades appear to be loosened from their frame. Every vertebra shows through the skin. The abdominal wall sinks in below the floating ribs and forms a hollow like a basin. Their thighs and the calves of their legs are reduced to a skeleton. One would say it was the picture of an immured nun, such as the old masters have portrayed. These women appear to be fifty or sixty years old. Sometimes they seem to be sustained by some unknown miracle of energy; their voices are strong and their steps firm. On the other hand, they often seem almost at the point of death, and ready to draw their last breath.

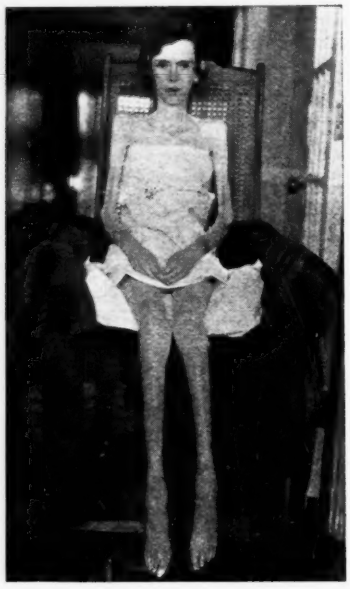

FIG. 1

FIG. 2

"Are they tuberculous or cancerous patients, or victims of muscular atrophies in the last stages, these women whom misery and hunger have reduced to this frightful gauntness? Nothing of the kind. Their lungs are healthy, there is no sign of organic affection. Although they look so old they are young women, girls, sometimes children. They may belong to good families, and be surrounded by every care. These patients are what are known as 'mental anorexics,' who, without having any physical lesions, but by the association of various troubles, all having a psychic origin, have lost a quarter, a third, and sometimes a half of their weight. The affection which has driven them to this point may have lasted months, sometimes years. Let it go on too long and death will occur, either from inanition or from secondary tuberculosis. However, it is a case of nothing but a purely psychic affection of which the mechanisms are of many kinds."

Weir Mitchell mentions that in its worst forms the patient develops such an aversion to food that spasm results from attempts to eat and if forced to eat often vomits. Several weeks may elapse without certain kinds of food which have previously been taken only on urgent persuasion. The skin is atrophied, dry and covered with bran-like scales; the patient lies on his side with the legs flexed on the knees and the thighs flexed on the trunk. Severe contractures and bedsores develop. The lower limbs are blue and cold.

FIG. 3

There is peristaltic unrest and diarrhea (Kussmaul), but usually marked constipation; pallor, with a high grade of anemia; slow weak pulse, and scanty urine.

Mentally he is irritable, restless, sleepless and obstinate with slow speech. In time, delirium and a slight fever may develop and the exhausted patient may succumb to the disease itself or to bacterial infection from loss of resistance.

Osler says that the emaciation with the marked retraction of the abdomen, sunken eyes and dilated pupils, is comparable only to that of cancer of the esophagus.

CASE 1.

Jean B. 25 years old, a white American girl in the upper social stratum, with a negative family history came under observation in 1916. There had been no serious illness and no emotional strain during her life. Her present illness was of four years' duration, beginning with gradual loss of appetite with progressive wasting but no loss of strength. In her daily life she could easily outdo her robust mother and sister. In 1912 her weight was 125, and height 70 inches. At the first consultation the information was volunteered that she "lived on coffee and cascara". Her weight at that time was 74. Amenorrhea had been total for one year. She presented, externally, the picture of far advanced tuberculosis but there was a complete absence of physical signs save the extreme emaciation. Jean had an exaggerated idea of her social obligations, spending her time as well as her allowance in the city slums, untiringly performing the most menial household tasks. Her relations with her family were strained, so she was persuaded to extend her vacation in New Hampshire.

Her weight, under treatment, went down to 68 lbs., but eventually, on a liberal diet, she began to gain and in three months had reached 90 lbs. Subsequently she regained her former weight and apparently completely recovered (1922). Marriage and pregnancy with normal delivery followed.

CASE 2.

Gladys F. 30 years old.

The first interview occurred on August 19, 1927.

Family History: Negative. Father, mother and brother living and well. Her family is in good circumstances and there have never been any emotional crises.

History of Illness: Well as a child. Until the age of twenty she lived a normal life. Operation for appendicitis was done in 1915 and a tonsillectomy in 1917. For no apparent reason she began gradually to take less food. She finished her training in a business school but made no effort to secure a position. In 1924 she was sent to a Boston hospital for study, but no diagnosis was made. Her weight continuously decreased, and went from 134 to 100 and in 1925 she began to stay in bed all the time. She conceived the idea that her father was unclean and forbade any contact between them. She felt the same toward her mother, but to a less degree. These delusions persist at the present time. She accepted food from her mother, though only after thorough scrubbing of the hands in her presence. She had no such fear of nurses, physicians, or other relatives. Twice each year she had developed pellagrous signs with symmetrical red scaling eruption on the dorsum of both hands and forearms and the anterior part of the neck; with beefy, red, sore burning tongue and mouth, and diarrhea. She lay in bed, on the right side with the legs flexed on the thighs and the thighs on the body. For years, except for short periods in a hospital, her food intake has been less than 100 calories per day, and for the greater part of the time almost totally vitamin free. There has been complete amenorrhea.

P.E. Many complete physical examinations have been made with practically no change in the findings except as noted. The hair is fine, silky and rather thin—eyebrows normal. The eyes are bright; cheeks deeply sunken—speech is slow and deliberate, with retardation when questioned. The thyroid is small but palpable; lymph glands normal. The muscles are atrophied and flabby. The skin is dry, thin and scaly. Bedsores have been noted at times, and though healing has been slow, they have ultimately disappeared. The ankles are constantly slightly edematous, the feet outstretched with markedly shortened tendo achillis. Toe nails are thick, corrugated and brittle. When pendant the feet rapidly become blue and cold, but rapidly return to color after pressure. The abdomen is sunken and boat-shaped. The heart and lungs are normal; the breasts, atrophied.

The blood pressure is not obtainable, the radial pulse scarcely perceptible. There is permanent flexion of the knees; the hip joints are not fixed.

Laboratory Findings. The basal metabolism is —12. Gastro-intestinal x-rays are negative. Radiographs of the bones show marked calcium absorption. The Wassermann test is negative. The blood sugar varies from 80 to 100 mgms. in each 100 c.c. of blood. The non-protein nitrogen, urea nitrogen, serum calcium, creatin and creatinin have always been in normal range of values. The blood morphology in average values shows hemoglobin (Dare) 65-80%, red cells 4,000,000 to 4,500,000; mean corpuscular volume 85 cubic microns, slight anisocytosis, and essentially normal percentages for various types of leucocytes. The urine is normal, many specimens having shown no suggestive changes.

Her weight measurements are:

1920	148
1927	68
1930	63
1931	58
1932	53
Present (May 13, 1932)	58

Progress. During the 5 years that the patient has been under observation very little change except progressive wasting has occurred. She had one attack of influenza from which she promptly recovered. At one time several bedsores developed following a transient urinary incontinence, but they healed. For short periods she has been in the hospital where she has always gained a few pounds in weight through semi-forced feeding, only to lose it promptly on her return home where she could perfectly manage a too sympathetic mother. Always the pellagrous manifestations cleared up with cod liver oil, orange or tomato juice and milk. On one occasion while in the hospital by means of screaming for a whole day she accomplished her purpose completely and a few hours later acknowledged that she had done so consciously.

At intervals thyroid extract has been administered, but on every occasion, after a short time there has been complaint of rapid heart, tremor and headache. In 1928 a tenotomy of the hamstring muscles in each leg was done and the subluxations partially reduced. This enabled her to walk comfortably with crutches with very little assistance. After a few months she gave this up, but motion in the knees has been preserved to the present.

Present Condition. Her weight is 53 pounds. She sleeps quietly all night. She attempts to refuse all food while at home, but under the care of a nurse, while in the hospital, she eats perfectly easily, taking 1500 to 1800 calories per day. At home she is up and dressed each day, and is carried by her brother to the most attractive room in the house. She reads, does embroidery and keeps up a large correspondence with members of a society of shut-ins. Constipation or other gastro-intestinal symptoms have not been present until recently when flatulence and occasional spilling occurred for a few weeks. A recent study showed the following measurements:

Calves of legs	7″
Thighs (mid)	9½
Forearms	5¾
Upper arms	5¼
Neck	10

From many conversations with this patient, and from observation the conclusion is justified that she has lost all desire to be physically attractive; that

she has lost all appetite and sense of hunger as well; that her persistent refusal of food is based on this condition rather than an actual determination to keep thin; or an actual belief that her mother is unclean. She calmly discusses these matters, expressing the conviction that her mental attitude is the easiest way to cease the annoyance of having repeatedly to refuse nourishment. She is perfectly comfortable in her present condition. To force food down her throat is followed by, to her, no little discomfort, amounting in some instances to actual pain and nausea.

Penzoldt has shown that hunger and appetite are separate terms though they have come to be used synonymously. "Hunger is the warning of food; appetite is the pleasure of partaking of it." Anorexia nervosa is described as a loss of appetite combined with a reduced or lost feeling of hunger. Loss of appetite in itself may be primarily a characteristic of hysteria or psychoneurosis. It may be secondary in gastroptosis or in diseases such as cancer or even ulcer of the stomach, where, because of pain the patient suppresses his desire for food.

We are considering here that condition in which the nervous or mental influence on the appetite seems mostly to be disturbed. In general the patients are women, though not all, and they are usually in an environment which tends to increase their interest in themselves and the value they place on their importance to society. The patient is not going to take any chances with this, his glorious position in the limelight. "He has a reputation to live up to," as Cameron puts it. (Leede.) Gull believed anorexia nervosa to be a nervous affliction, involving especially the pneumogastric. The name has been used to denote the anorexia of hysteria, though the stigmata of hysteria are usually wanting, except the mental phases.

While some writers affirm that anorexia nervosa always follows some emotional strain or prolonged nervous excitement, there is evidence that the neurotic, hysterical, neuropathic patient can by a mere whim develop into a state where, as Paul Dubois says "all feminine coquetry has disappeared, and they admit, without being in any degree impressed by it, their paleness and the fact that they have wasted away to skeletons". Whether there is a coexistent endocrine metabolic basis for this truly awful picture is as yet unproved. Berkman states that exhaustive study of a large group of cases shows nothing that will explain the symptoms or physical condition of these patients.

Nearly every group of patients reported contains a preponderance of women. The average age falls between twenty and forty years. The literature contains reports of small series of cases. Gull, in his original paper described three, all of whom recovered, one in six years, one in two years, and one in one year.

Comby in describing four cases first suggested that there must be some underlying endocrine disorder, since he noted that amenorrhea preceded the anorexia. Jelliffe mentions that no organic lesions were shown at necropsy. Osler describes one such case, in which he performed the autopsy, where the subject weighed 49 pounds. Venables concludes that bad mental states have little to do with the etiology. He affirms that recovery occurred in all his nine cases, and that all investigations (cardiovascular, respiratory, accessory sinuses, as well as chemical, microscopical and cultural examinations of the stools, urine and blood), showed normal conditions. Gastro-intestinal radiographs and study of gastric chemistry were not suggestive of any change. Leede found a low basal metabolism, low blood sugar, and evidence of glandular hypofunction, particularly hypoadrenia.

Berkman, in the largest series yet reported (117 cases) concludes that anorexia nervosa is not a common disease; that the low basal metabolism is due to the inanition; and that reëstablishment of normal food intake in this physiologic disorder which is second to a psychic disturbance should be the goal; and that thyroid gland substitution in small doses continued for a long period is a valuable measure.

The diagnosis of anorexia nervosa can be made only after the careful exclusion of organic disease. The difficulty appears to be to recognize the patient who has a true mental anorexia in the process of development. Many of these cases go unrecognized and untreated for years.

The disease is serious in that it lowers the patient's resistance; and mental anorexia may of itself cause death. Some patients have been known to lose half their usual weight, when Dejerine says no treatment will save them. The patient who is cured apparently recovers completely, but relapses occur in cases that are badly treated, and in these instances loss of weight is much more rapid than in the first attack.

Apparently all authorities are in agreement that change of environment, psychotherapy and forced feeding are the essentials of management. From personal experience with the cases reported herewith the writer can sincerely endorse the postulates of Leede; that so long as the patient holds the center of the stage in a family too easily given to sentiment, instead of reason, very little can be accomplished in rescuing the victim from his pitiable plight.

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2 Jelliffe, Smith Ely: Osler's Modern Medicine. 5: 163, 1908.

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4 Venables, John F.: Anorexia nervosa, study of pathogenesis and treatment of nine cases. Guy's Hosp. Rep. 80: 213 (Apr.) 1930.

5 Leede, C. S.: Anorexia nervosa, hypoglycemia or hypoadrenia. Northwest Med. 27: 233 (May) 1928.

6 Berkman, John M.: Anorexia nervosa, anorexia, inanition and low basal metabolic rate. Am. J. M. Sc. 180: 411, 1930.

Discussion

Dr. Harry T. French, Hanover: I want to thank Dr. Clow for his admirable presentation of this subject. This question of appetite comes into the picture in many cases that are not so definite as anorexia nervosa. I was interested in his differentiation of hunger and appetite. Perhaps if we should analyze hunger it would depend largely upon muscular contraction of the stomach, while appetite is a much more complex matter, involving psychic factors and certain physical features, such as distress in the gastro-intestinal tract, the gastric secretion, the level of the blood sugar, the condition of the thyroid, and hypothyroidism or hyperthyroidism.

As Dr. Clow pointed out, anorexia nervosa may be divided into primary and secondary, the secondary, largely dependent upon some physical factor present. There has been some very interesting work done recently in connection with animals in regard to the loss of appetite. This has shown that dogs fed on a diet free from vitamin B, develop a marked loss of appetite, absolutely refuse to eat, and apparently go through a condition similar to that which is called anorexia nervosa, but if those dogs are given a fair amount of vitamin B, the appetite is certain to return and if large amounts are given, the loss of appetite is corrected and return to normal condition follows.

Anorexia develops frequently with psychic shock, and that because of the mental state no food is taken for a time lowering the level of vitamin B in the body thereby developing a vicious circle. I wonder whether if high vitamin B feedings were attempted in some of these cases they might not produce some result. The basal metabolism in some of these cases runs low, and some degree of improvement follows the use of thyroid extract. In view of the remarkable results which have been obtained by the use of insulin in very emaciated patients suffering from tuberculosis I should think its trial would be worthwhile in this type of case.

It is interesting, in connection with Berkman's work, that the low metabolisms found in his study of this series of cases cannot be blamed on the inanition as it has been shown that inanition of itself does not produce low metabolism rates.

Gastric secretion or lack of gastric secretion frequently in children is responsible for loss of appetite. We know if we stimulate the gastric secretion we find that there is a return of appetite.

I would like to ask Dr. Clow if he has by any chance in these cases tried insulin or high vitamin B feeding?

Dr. Clow, closing: It so happens that in our case the economic factor is important. The patient is not able to have hospital care for a long enough period to really accomplish very much. Gladys F., has been hospitalized five times for a short period, and she always gained in weight. In 1931 we used insulin in small doses, and she gained her usual amount in the much shorter time that she was there, about the same as she had in previous years. We feel confident, that if provision were made so that this girl might be kept under treatment for months instead of weeks, she could probably be brought back to normal. However, Dejerine in his book, says he never has seen a patient who lost more than half his weight, come back.

RECENT DEATH

THURBER—Madison T. Thurber of Boston, Mass., 30 Thornley Street, an affiliated member of the New Hampshire Medical Society for a number of years, died at his home September 27.

See page 633 for extended notice.

CONCLUSIONS RESPECTING THE COST OF TREATING SYPHILIS

By Michael M. Davis, Ph.D.

Estimates of the proportion of the population who can pay any specified amount for medical service are necessarily subject to a considerable margin of error. Nevertheless it has seemed important and justifiable to formulate in quantitative terms the writer's judgment as to the significance of the facts presented in this article. The percentages stated in the two following sentences are the writer's opinion of the most probable figures.

(1) About 80 per cent of the population cannot pay for adequate care of syphilis at private rates.

(2) About 50 per cent of the population can pay for the adequate care of syphilis at pay clinic rates, although not at private rates; except when the usual minimum fee is cut by the physician to about half.

(3) Fully one-third of the population, if infected, must receive treatment for syphilis free or for nominal amounts.

(4) The cost of treating syphilis is an important cause of the insufficient or inadequate treatment which many infected persons receive. — Reprinted from *Journal of Social Hygiene*, October, 1932, Julius Rosenwald Fund, Chicago.

VERMONT STATE MEDICAL SOCIETY

THE ONE HUNDRED AND NINETEENTH ANNUAL MEETING OF THE VERMONT STATE MEDICAL SOCIETY

The One Hundred and Nineteenth Annual Meeting of the Vermont State Medical Society will be held at the College of Medicine, University of Vermont, Burlington, Thursday and Friday, October 6 and 7, 1932.

Officers for 1931-1932

President—E. J. Rogers.
Vice-President—A. M. Cram.
Secretary—Wm. G. Ricker.
Treasurer—David Marvin.
Auditor—E. H. Buttles.

Councillors—1st District: Alan Davidson, St. Albans; 2nd District: John Trotter, Bennington; 3rd District: F. E. Farmer, St. Johnsbury, 4th District: John Woodruff, Barre.

Executive Committee—Wm. G. Ricker, C. G. Schurman, O. N. Eastman.

Publication Committee—Wm. G. Ricker, C. F. Dalton, E. A. Stanley.

Legislative Committee—John Stevenson, L. H. Ross, R. M. Pelton.

Medical Education—A. C. Black (1), S. W. Hammond (2), John H. Woodruff (3).

Necrology Committee—George G. Marshall, B. D. Adams, H. L. Pierce.

Medico-Legal Committee—J. H. Jenne (1), F. E. Farmer (2), E. A. Hyatt (3).

Health and Public Instruction Committee—E. H. Buttles, B. F. Cook, P. K. French, A. L. Patch, E. H. Ross.

Delegates

Maine—T. S. Brown.
New Hampshire—C. F. Ball.
Massachusetts—C. H. Swift.
Connecticut—E. A. Tobin.
Rhode Island—C. D. Rublee.
New York—G. M. Sabin.
A. M. A.—Wm. G. Ricker.
Alternate A. M. A.—C. H. Beecher.
Anniversary Chairman—C. H. Beecher.

New England Medical Council

E. J. Rogers, Wm. G. Ricker, W. M. Johnstone, J. A. Stevenson, E. A. Hyatt.

Local Committee of Arrangements

Sam Sparhawk, Chairman, H. E. Upton.

Ladies' Committee

Mrs. H. E. Upton, Chairman, Mrs. C. H. Beecher, Mrs. Sam Sparhawk.

Officers of the House of Delegates

President—Frank E. Farmer, St. Johnsbury.
1st Vice-President—Seth Martin, Burlington.
2nd Vice-President—P. L. Dorey, Middlebury.
Secretary—Naomi Lanou, Rutland.

PROGRAM

Thursday Forenoon, October 6, 1932, at Nine O'Clock, at the College of Medicine, University of Vermont

1. Call to Order by the President, E. J. Rogers, M.D., Pittsford.
2. Prayer, Rev. Skillman E. Myers.
3. Address of Welcome, Sam Sparhawk, M.D., Burlington.
4. Reports of the Officers and Committees.
5. Reception of the Delegates from other Societies.
6. Vice-President's Address, Albert M. Cram, M.D., Bridgewater.
   Title: "Diagnosis and Treatment of Trigeminal Neuralgia."
7. Paper, Charles Armstrong, M.D., Surgeon, U. S. Public Health Service, Washington, D. C.
   Title: "The Diagnosis of Mild Smallpox and of Scarlet Fever."
8. Paper, Richard B. Cattell, M.D., Surgeon, Lahey Clinic, Boston. Assistant Surgeon, New England Deaconess Hospital and New England Baptist Hospital.
   Title: "Carcinoma of the Rectum."
9. Announcements by exhibitors of Scientific specimens.
10. Address, Edward H. Cary, M.D., President of the American Medical Association, 1931-1932, Dallas, Texas.
    Title: "Ophthalmic Diagnosis."

Thursday Afternoon at Two O'Clock

11. President's Address, Edward J. Rogers, M.D., Supt. and Medical Director Vermont Sanatorium, Pittsford.
    Title: "Pneumothorax Treatment in Tuberculosis." Practical Demonstration of Pneumothorax Treatment.
12. Paper, Edward D. Churchill, M.D., John Homans Professor of Surgery, Harvard Medical School, Chief of the West Surgical Service, Massachusetts General Hospital, Boston.
    Title: "Non-Tuberculous Pulmonary Suppuration."
13. Paper, Donald S. King, M.D., Chief of Pulmonary Clinic, Massachusetts General Hospital, Boston.
    Title: "Tumors (Benign and Malignant) of the Lungs, Bronchi and Mediastinum."
    All speakers will use x-ray films.

FIXED SESSION OF THE HOUSE OF DELEGATES, 5 O'CLOCK, COLLEGE OF MEDICINE

List of members, reports of the committees and order of business are printed in pamphlet form. Copies may be obtained at the registration desk.

THURSDAY EVENING, AT SIX-THIRTY O'CLOCK, AT THE ETHAN ALLEN CLUB

The Annual Banquet.

Anniversary Chairman, C. H. Beecher, M.D., Burlington.

FRIDAY FORENOON, NINE O'CLOCK, AT THE COLLEGE OF MEDICINE, UNIVERSITY OF VERMONT

14. Report of the House of Delegates.
15. Symposium. "Obstetrics."
    "Ante-natal Care"—Ivan Y. Patrick, M.D., Associate in Gynecology and Obstetrics, Royal Victoria Hospital, Montreal.
    "Normal Delivery"—James W. Duncan, M.D., Clinical Professor of Obstetrics, McGill University, Montreal.
    "Surgical Delivery per Vagina"—Gerald C. Melhado, M.D., Associate in Obstetrics and Gynecology, Royal Victoria Hospital, Montreal.
    "Surgical Delivery, Supra-pubic"—Herbert M. Little, M.D., Professor of Obstetrics, McGill University, Gynecologist to the Montreal General Hospital, Montreal.

ENTERTAINMENT

A luncheon will be given for the visiting ladies at the Klifa Club, 342 Pearl Street, Thursday at one o'clock.

Luncheon for physicians at the Medical College Thursday from twelve to one o'clock.

Following the banquet Thursday evening, dancing, bridge, pool, billiards and bowling may be enjoyed at the club house.

NOTES

The Headquarters of the Society and Registration Desk will be at the College of Medicine, University of Vermont, during the hours of sessions.

Banquet tickets should be purchased when registering at the Medical College.

There will be a scientific exhibit and a commercial exhibit in the laboratories at the College.

HOTEL RESERVATIONS

Reservations of rooms can be made at and through the hotels, Vermont, Van Ness and Sherwood.

BY-LAWS

Article IX—Time for Papers

No author shall consume more than twenty minutes in reading or presenting a paper and no one shall speak more than five minutes in the discussion of a paper, without unanimous consent of the members present.

Article X—Addresses Property of Society

All addresses and papers presented in the County and State Societies thereby become the property of the State Society, and shall be placed in the hands of the Secretary within one week after the meeting, for insertion in the Records.

Article XI—Order of Business

The program as prepared by the Executive Committee and published in accordance with the By-Laws by the Secretary shall constitute the order of business, and can not be changed, or suspended, except for a definite purpose, a limited time and by a two-thirds vote of the members present.

VERMONT DEPARTMENT OF PUBLIC HEALTH

AUGUST, 1932

Communicable diseases reported during the month of August are as follows: chicken pox 25, diphtheria 4, measles 28, mumps 100, poliomyelitis 1, scarlet fever 25, tuberculosis 9 and whooping cough 30.

The total number of examinations made in August by the Laboratory of Hygiene was 1719, classified as follows:

Examinations	
For diphtheria bacilli	74
For Widal reaction of typhoid fever	26
For tubercle bacilli	161
For evidence of syphilis	471
For gonococci in pus	159
Of water, chemical and bacteriological	200
Of water, bacteriological	215
Of milk, market	178
Of milk, submitted for chemical only	13
Of milk, submitted for microscopical only	132
For the courts, autopsies	5
For the courts, miscellaneous	20
Of animal heads for evidence of rabies	3
Miscellaneous	61
Autopsies to complete death returns	2

Sixty-seven cases of gonorrhea and thirty-two cases of syphilis were reported to the Division of Communicable Diseases. Three hundred and sixty-six Wassermann outfits and 241 slides for gonorrhea were distributed.

A total of 558 patients was seen at the summer clinics by our staff of workers in the Poliomyelitis After-care Division and Doctors Frank R. Ober and Joseph S. Barr of Boston. These clinics were held at nine centers in the State. Thirty-nine home visits were made by the nurses, 14 new pieces of apparatus fitted and 48 corrections made to shoes for patients. The vocational worker in this Division reports sales made amounting to $355.98, five sales being held at different points.

The State Advisory Nurse of the Maternity and Infancy Division has assisted in several preschool clinics as well as preliminary clinic meetings in five towns. Three hundred and thirty-four notifications of birth registration and 799 pieces of literature have been sent out.

MEDICAL PROGRESS

PROGRESS IN ANESTHESIA IN 1931

BY RUSSELL F. SHELDON, M.D.*

A REVIEW of articles brought out on any subject in any given year makes uninteresting reading, and no attempt to list such a "catalogue of the ships" will be made. In the 1931 report of the "Progress of Anesthesia in 1930" Woodbridge[1] has so well brought the subject to date that it is difficult to draw a 1931 picture without some repetition. However, like him, the present reviewer will indulge in some personal comments. When one reads the 1932 Anesthesia section of one of the "best sellers" in Medical Encyclopedias, however, and finds it a virtual summary of Woodbridge's work, it is easily noted that the standard he has set is a high one. Organized anesthesia is truly fortunate, for as its leader it has an untiring enthusiastic individual, ably and cheerfully assisted by an equally untiring enthusiastic wife. I refer, of course, to Dr. and Mrs. Frank McMechan of Rocky River, Ohio. Their whole life is in the specialty, and for all its members, they are true friends and coworkers. As a matter of cold reality, a report of the Progress of Anesthesia should be extremely easy, as a complete bibliography is maintained by the McMechans and brought up to date every other month in the journal *Current Researches in Anesthesia and Analgesia.*

Furthermore, in 1931, appeared a most complete survey of the field of anesthesia, a masterpiece, of which a verbatim transcript would amply fulfill the fondest desires of the editor. The Fiske Fund prize essay, No. LXVI[2], entitled "Anesthetics, their relative values and dangers" by Dr. Albert H. Miller of Providence, R. I., is in itself a complete course in anesthesia, which every student and teacher should find invaluable. By its award Dr. Miller was enriched to the extent of two hundred dollars, but one who neglects this work has forfeited many times that amount.

The search for the perfect anesthetic continues, but the present trend[3] is less toward a single "knock out" preparation than to a combination of those agents already in use, providing to the patient an experience in surgical and operating-room technique that he would be glad to repeat. In other words, efforts are being directed to the comfort, as well as to the safety, of the patient; and to prove false the old concept: "I don't mind the operation, but I dread the anesthetic." Now in 1932, one frequently hears "The anesthetic is the one part of the operation I look forward to." Of course this is truer for those requiring a second or additional administration, but their testimony is very helpful and encouraging to those undergoing for the first time the experience of a surgical operation. Certainly there has been great progress in making the operating-room "approach" less formidable.

The desideratum of the surgeon is a foolproof anesthetic that will in itself convert a bad risk into a good one. Much as an anesthetist hates to disappoint a surgeon he at once admits his failure to meet this desire. By use of selected methods and drugs he tries to coöperate with the surgeon, lessening the degree of risk, but not entirely eliminating it. "Balanced Anesthesia[4]" the title of an address by Dr. John Lundy of the Mayo Clinic, is a concrete example of what has been accomplished by the combination of agents already known, to produce a satisfactory experience for the patient subjected to surgery. As he so well points out there is no drug or method 100 per cent desirable for every case. Small doses of two or three agents in safe limits are far preferable to a single large dose, especially in "bad risk" patients, and the method, as well as the drug, is subject to change, as comfort and safety demand.

One of the strongest factors in welding together a group with similar interests is the annual convention. Not that any startling disclosures were announced at the 1931 convention in New York, but a very spectacular demonstration was given by Waters of the extension of his intratracheal method by the Guedel-Waters[5] balloon (inflatable cuff in endotracheal catheter) to render it intrabronchial, thus making it possible to block off one lung. For the thoracic surgeons* this makes a tremendous advance for their anesthesia.

A noteworthy feature of an anesthesia convention is in the mingling not only of physicians interested in the specialty, but of the manufacturers who produce both the drugs used and the apparatus by which they may be administered. One result of this coöperation, not however suggested in 1931, but brought to fulfilment in that year, is the uniform color of cylinders containing anesthetic gases, regardless of the manufacturer. All nitrous oxide tanks are now blue, oxygen green, carbon dioxide gray and ethylene red. This spirit among the manufacturers has eliminated much confusion and has undoubtedly been a factor in saving lives.

*Sheldon—Assistant Anesthetist, Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary. For record and address of author see "This Week's Issue," page 632.

*See Coryllos, P. N.: A method of combined endotracheal anesthesia and suction in thoracoplasty for pulmonary tuberculosis. Results in 41 cases (79 operations). Current Researches in Anesthesia and Analgesia. 11:138 (May-June) 1932.

One development of the 1931 convention was the introduction of machinery eventually to provide an international college of anesthetists similar in ideals to the colleges of surgeons and physicians. Anesthesia is already organized internationally, but with the perfection of the newly conceived college, a patient, anywhere in the world, would have at his disposal facilities for ascertaining the standing, not only of his surgeon or hospital, but also of his anesthetist.

Of new apparatus brought out in 1931, the new Connell machine is outstanding. Although far from simple in appearance, it embodies safety, in freedom from explosions by the use of water, "Free wheeling," by the use of Waters' carbon dioxide filter, ease and visibility of control, plus the fundamental quality of "taking you there and bringing you back." Its size and lack of portability make it available for use only in institutions, and not for the individual anesthetist.

In an effort to show that ethylene is our best single anesthetic, Bevan[6] sets up a list of standards for the judgment of anesthetic drugs. These are

1. Safety of patient.
2. Comfort.
3. Efficiency of anesthetic agent.
4. Control.
5. Elimination of psychic trauma through abolishing both pain and consciousness.
6. Simplicity and general adaptability of method.
7. Absence of injurious effects on organs, tissues or blood.
8. Absence of complications—vomiting, headache, delirium.
9. Absence of effects that lower resistance to organisms, such as pneumococci and streptococci.

The strongest feature is the production of a series of 20,000 cases without a death.

While all authors unite in minimizing the danger of explosion with ethylene, Miller[2] summarizes thus: "The chance of explosion of mixtures of ethylene and oxygen acts as a deterrent to its more extensive use. It is not the number, but the nature of these explosions, which is considered. One who would pass away quietly in bed without protest, revolts at the idea of violent death from intra-pulmonary explosion."

In Montreal, where winters are proverbially severe, Griffith[7] finds no contraindication to the year-round use of ethylene. By keeping the relative humidity of the operating room at 54 per cent or higher he has no fear of explosion, and he describes his experience with a device called the "zephyr" which at an installed cost of $120 approximately, has kept his operating room at a safe degree of humidity.

Basal anesthesia is a term much to the fore in modern anesthesia literature. Preliminary medication has, indeed, been with us for a long time, and no proof of its desirability and helpfulness is needed. Woodbridge[1] defines basal anesthesia as "so deep a state of narcosis produced by preliminary medication that the amount of added inhalation anesthetic required to produce surgical anesthesia is greatly reduced, and the patient is unaware of the administration." In other words, basal anesthesia is merely our old friend preliminary medication considerably dressed up. With this understanding we find little to add to the 1930 status. Some discussion is always in order, however. Morphine and scopolamine have in many cases amply fulfilled Woodbridge's definition. Some individuals have gone through surgery with sodium amytal (or other barbiturate) alone and certainly with the barbiturates, a less quantity of general anesthetic is required. But it is with the rectal instillation of tribromethanol (Avertin) that we find the greatest use of the term "basal anesthesia". With this drug a state closer to true anesthesia is reached, as there is more complete muscular relaxation, and its duration may be long. Far more than sensory inhibitive effect on the nervous system is produced by this drug. There is indeed an "intoxication", not dissimilar to that produced by alcohol, for the drug is an alcohol derivative. Patients get "drunk" with avertin and complete amnesia follows. A hangover is very rare; but with scopolamine and the barbiturates the hangover may assume violent proportions.

One of the most interesting articles[8] on this new rectal "anesthetic" comes from Edinburgh with a discussion on the fall of blood pressure in a series of gynecologic operations. The comparison is made between the patient on the operating-table and the "subject" in a Turkish bath. Both are relaxed and undergoing no voluntary exertion. The observation is made that the latter undergoes a considerable drop in blood pressure with no deleterious effect, in fact with a feeling of benefit; and no harmful results have occurred to the surgical patient in his series with a similar drop in blood pressure. Kennedy feels therefore, that in some individuals at least, a fall in blood pressure may not be harmful. But we do know that, in general, patients stand a rise in blood pressure better than a fall. In fact, the blood pressure drop in spinal anesthesia is a deterrent in many cases. It is well known, even among the laity, that persons with "weak hearts" should avoid Turkish baths. Is the fall in blood pressure the answer? Perhaps a more scientific study of Turkish baths will give additional light on one of the problems of anesthesia.

In this country, distilled water is ordinarily used as a vehicle for instilling avertin. Kennedy, however, uses tap water. In about half of his patients headache occurs postoperatively.

This is a greater incidence than reported by American authors, though it is hardly possible that tap water can be the cause.

McKittrick, McClure and Sweet[9] give an accurate presentation of the use of *spinal anesthesia* in abdominal surgery. This is a straightforward report of their own results, and is a sensible and conservative contribution to the long list of articles on that subject. They make no extravagant claims, and their conclusions are sound. They say: "Spinal anesthesia does not fulfill all the requirements essential to the ideal anesthetic for abdominal surgery. Nevertheless it has an important place in surgery below the diaphragm. Its advantages are complete relaxation, quiet respirations, contracted intestine, a probable lack of direct irritation to the kidneys, liver and lungs and the absence of any direct effect on the metabolism of a patient with diabetes mellitus. The disadvantages are a short uncertain period of anesthesia, the uncertainty of its effect on blood pressure, occasional nausea and vomiting occurring during operation, and the slight handicap of working in the upper part of the abdomen with the patient in Trendelenburg position."

Of somewhat similar nature are the conclusions reached by Muller and Overholt[10] in Philadelphia.

1. Spinal anesthesia carries with it a higher table mortality than other forms of anesthesia in our hands. Its many advantages often outweigh the added risk that it carries, and for that reason spinal anesthesia has become a valuable adjunct to our present-day anesthetic methods.
2. Hypertension, hypotension or any condition which will in itself produce a hypotension during the course of operation, constitutes a contraindication to the use of spinal anesthesia.
3. Open drop ether in the poor risk patient is the safest anesthetic. Spinal anesthesia is reserved for the good risk patient where the added danger of the anesthesia can be assumed in order to facilitate the technical operative procedure.
4. Sudden deaths after operation should be charged up partially against spinal anesthesia when the hypotension induced by this form of anesthesia persists.
5. The site of the operation or the dose of the drug has a surprisingly small influence upon the degree of the fall in blood pressure, or in the time in which the maximal fall takes place.
6. The incidence of pulmonary complications is not reduced by spinal anesthesia.

Rapoport[11], with a series of 1875 cases, also gives a clear concise picture of the present use and status of spinal. He states that the only contraindication to spinal is a moribund patient. It might be a question whether surgery itself is desirable under such a condition. "Patients with hypertension and hypertensive heart disease furnish the largest number of bad reactions. The drop that spinal anesthesia causes in the blood pressure is directly related to its height before operation. The higher the blood pressure the greater the drop."

Early in the year, after a report by Lundy[12] on the use of *pantocaine* in 400 cases it seemed that a safe substitute for novocaine, eliminating the uncertain time element of the latter drug, had appeared. "Pantocaine is effective in 1/5 to 1/10 the dose that is required if procaine is used and its effect lasts about two to three times as long, depending on the size of the dose. It is three to four times less toxic than nupercaine." Further pronouncements of its efficacy and freedom from toxicity however have not appeared, and in fact at the convention in October this "vacuum" was definitely noticeable. Throughout the country the solution of novocaine crystals in spinal fluid is the usual method for spinal anesthesia.

"Procaine (Novocaine) for about 25 years has been acknowledged to be the safest local anesthetic agent, and in spite of demands for an agent, the effect of which will endure longer, none has been brought out that has been in use for a sufficient time to have been proved to be a success clinically."

A simple quantitative test for the efficacy of local anesthetics was announced from the Mayo Clinic[13], using the usual laboratory frog. By injection intracranially or intraspinally, through a cisternal puncture, measured doses of various drugs can be given, and the length of time the frog remains limp is easily noted. Care must be exercised to prevent inserting the needle into the brain or spinal cord. In non-fatal doses the same frog may be repeatedly used.

Among the most careful attempts to solve the problem of what happens in spinal anesthesia may be mentioned articles by Lundy and Essex[14], and Seevers and Waters[15]. The fall in blood pressure is studied and the use and action of ephedrine observed.

Seevers and Waters conclude:

1. Cellular oxygen want per se is one of the prime factors in circulatory depression accompanying spinal anesthesia.
2. Decrease in the oxygen content and increased CO_2 content of the blood is present when the blood pressure is low accompanying spinal anesthesia*.
3. Under high spinal anesthesia, with some degree of intercostal paralysis, the circulatory condition is improved when oxygen rich air is inhaled.
4. The fall of blood pressure in spinal anesthesia tends to be synchronous with and pro-

*This is not true in the fall of blood pressure accompanying Avertin Anesthesia. See Taylor, G. W., and Lund, C. C.: Blood pressure fall occurring in avertin anesthesia. New Eng. J. Med. 206:612 (Mar. 24) 1932.

portional to the amount of intercostal muscle paralysis produced.

5. Efficient two phase artificial respiration will maintain normal blood pressure in presence of cord section at the seventh cervical segment, or novocaine motor and sensory root paralysis of the whole central nervous system.

6. Under high spinal anesthesia, even when blood pressure has been sustained by ephedrin, there is hypersensitivity to anoxemia. No primary asphyxial rise in systolic pressure occurs.

7. Ephedrin in varying dosage tends to maintain normal blood pressure when administered previous to induction of high spinal block.

8. After the blood pressure drop in high spinal anesthesia, ephedrin is less prompt and less effective if administered by other than intravenous injection.

9. When low blood pressure is present from causes other than spinal paralysis, such as extensive hemorrhage or prolonged anoxemia, ephedrin may fail to restore blood pressure even when given intravenously.

10. High oxygen content of the blood augments the beneficial effect of ephedrin.

11. We have not yet seen evidence of circulatory or other damage from ephedrin if the dose is limited to that sufficient to restore normal blood pressure provided the tissues are adequately oxygenated.

12. Cord section at the fifth thoracic segment, or complete novocaine paralysis below this point produces no marked change in blood pressure in the normal dog.

13. With our present knowledge, treatment of accidents following spinal anesthesia should consist of two phase, artificial respiration (preferably with oxygen) plus intravenous ephedrin in such doses that blood pressure is maintained at the pre-anesthetic level. Existing fluid deficit should of course be made good.

14. Circulatory depression following high spinal anesthesia with intercostal paralysis is much more marked in animals anesthetized with barbital, ethylene, nitrous oxide, or having morphine-scopolamine as a pre-operative sedative, than in the unanesthetized animal.

In a careful attempt to show, if any, the harm done to the cord and its membranes by spinal anesthesia, Davis[16] and his co-workers in Chicago reported a painstaking piece of work on dogs, using nupercain and other novocaine derivatives. Except for the immediate effect of trauma little evidence of permanent damage was found; in fact, the animals killed after 90 days showed very little pathology. Certainly the objectors to spinal anesthesia can find little pathological evidence that the process is harmful, but further study on human beings is to follow.

CO_2 Absorption[6]. One of the results of economic depression has been an increased desire among hospital administrators to save money. Any method of conservation therefore has this year received additional support. In spite of many advantages claimed by Waters[17], such as smoother maintenance, less loss of body heat, etc., in previous years the chief advantage of his "Carbon Dioxide Absorption Technique" has been proclaimed by others to be its economy. For hospital executives, or anesthesia departments, no better "Manual" for the introduction of this technique has appeared than Waters' article in *California and Western Medicine**. If one has not previously been "sold" on the merits of this system, perusal of this will surely convince him.

Any method which saves from 30% to 60% (conservatively) of gases naturally does not have the spontaneous support of the gas manufacturer, and the apparatus manufacturer quite naturally dislikes to antagonize the gas manufacturer. There is, therefore, very little active salesmanship to bring the CO_2 filtration equipment to the notice of hospitals and anesthetists. I cannot too strongly urge the careful study of Waters' publication.

In the British possessions *intratracheal* (with accent on the penult) methods have had a greater vogue than in this country. Even our Canadian friends have adopted this procedure to a considerable extent. The year 1931 marks its large scale introduction into Boston with its use by Sise and Woodbridge at the Lahey Clinic. There has also been increased use at the Mayo Clinic.

Therapeutic Use. Everything from angina pectoris[18] to ringworm of the feet[19] has been cured by anesthetic agents. Of chief interest is the further use of oxygen tents, which are now supplied by the leading gas manufacturers and supply houses and transported anywhere at short notice. In hospitals there has been further introduction of the Drinker respirator, and trained personnel for all inhalation therapy has become more available. For treatment in the home however, inhalation therapy is considerably handicapped by its cost, not only for the gases used and the apparatus for administering them, but for the personnel. Trained anesthetists (medical or nursing) are expensive, particularly if their services are required over long periods, as in the treatment of pneumonia by oxygen. However, the taxpayers are unwilling that deserving patients be deprived of this modern facility, and have supplied not only material and apparatus, but also the personnel. Most recipients of aid from the public treasury are not anxious for publication of such fact, but there is something spectacular about the practice of inhalation therapy by the Fire Department, and the papers duly record it. In most accounts, aid of the public authorities has been requested

*Reprinted in Current Researches in Anesthesia and Analgesia. 11:97 (May-June) 1932.

by the attending physician, who of course is entitled to all the help he can get from anybody. Discussion or comment on State Medicine is far beyond the scope of this review, however. We merely record that there has been an extension of this practice during 1931.

It has been previously shown in animals that the primary toxic action of *propylene* is upon the respiratory center, and secondarily upon the heart. With Riggs as the subject[20] of propylene anesthesia two or three times a day, with weekly intervals to permit the dissipation of any possible overhanging events from one period of anesthesia to the next, the same conclusions as in animals are reached in man. "Propylene used as a general anesthetic in man produces complete anesthesia after an average of 8 inhalations. There occurs no struggling or any reactive symptoms during the period of induction. During complete anesthesia the breathing is quiet and regular." This is an interesting study, but any drug with a "primary toxic action on the respiratory center" is hardly acceptable for safe general anesthesia.

Obstetrical anesthesia is and probably always will be a fertile field for discussion. Gwathmey's ether-oil mixture by rectum seems much more favored by those doing obstetrics in the home, than by those with hospital practice. In the latter there is marked increase in the use of the barbiturates, with or without rectal ether. In hospital practice gas oxygen at the time of delivery is nearly universal. Of eight speakers at the 1931 meeting of the Section of Obstetrics of the Massachusetts Medical Society[21], Gwathmey's technique was mentioned by two and favored by one, that one being a woman, but nearly all based their remarks on hospital rather than home obstetrics.

The subject of *postoperative pulmonary complications* is proving a surgical much more than an anesthetic problem. The study of King[22] soon to be published, but in part reported at a clinical meeting at the Massachusetts General Hospital, has demonstrated that in a large series of these cases the anesthetic is a minor factor. In one of the best reports on this subject the author[23] makes no mention of anesthesia, and as a surgeon he accepts the responsibility of prevention and care. The use of carbon dioxide in prevention has been disappointing, as demonstrated by King[22].

In continuation of Rowe's work, Mendenhall and Connolly[24] made a study of the action of the cilia of live oysters with pure and contaminated ether. This is a remarkable series of experiments carried out under extreme technical difficulties. With peroxide-aldehyde contaminations .01 to .02 per cent definite inhibition of ciliary action was observed; with pure ether, effect on ciliary action was negligible or slight. If the same condition holds true in mammalian cilia, ether impurities may be a factor in the causation of bronchial plugs, and secondarily of atelectasis and collapse, and pneumonia. Due, however, to the care used by manufacturers both in the process of manufacture and in the containers in which ether is marketed, and in addition, to the supervision by federal, state and municipal authorities, the causation of pulmonary complications by aldehydes and peroxides in commercial ether is today almost beyond consideration. It is largely due to the pioneer work of Rowe that this happy condition has been reached. By way of warning, however, it must be emphasized that when ether is used, it should be fresh. The danger of using ether which has stood exposed for some time, as in the glass container on a gas machine, is shown by this experimental study to have a real physiological basis, and the avoidance of the use of old ether is a factor in the prevention of postoperative pulmonary complications.

In conclusion, there is no better statement of the present situation in anesthesia than that given by Miller[2]. "If we knew but one of the anesthetic agents, this one would be considered a priceless boon, and would be extensively used, whatever its inefficiency and danger. A like condition can be found at the present time in many institutions and communities where one routine anesthetic, ether, nitrous oxide, ethylene, regional or spinal anesthesia is used to the exclusion of others. . . .

"The only method which has stood the test of time is ether inhalation, which since its introduction by Morton, has never failed to be utilized for overcoming the shortcomings of other methods. Ether inhalation occupies the position of the most extensively used method of anesthesia in spite of great handicaps. Millions of dollars have been invested in accurate measuring apparatus for use with nitrous oxide and ethylene. For regional and spinal anesthesia a highly developed technic is available.

"Except for the continuous process devised by Squibb for manufacture of the agent, the work of Boothby and Connell on measured dosage, the oil ether colonic method of Gwathmey, and the introduction of insufflation, no noteworthy improvement over Morton's method has been made since its origin, eighty-five years ago."

The present reviewer would respectfully add to the above exceptions Henderson and Sword, for the introduction of carbon dioxide as a respiratory stimulant and Waters, for the introduction of "free wheeling" into anesthesia by taking from the expired air carbon dioxide by his soda lime "filter".

"The value of an anesthetic depends upon its safety, its efficiency, and the degree of comfort afforded the patient. Chloroform is probably the most efficient anesthetic but its immediate toxic action on the heart and its delayed poisonous effect put it on the danger list. Ethyl Chlo-

ride provides an ideal induction to less agreeable anesthetics, but is inferior to nitrous oxide in safety.

"Statistics of long series of successful nitrous oxide oxygen administrations prove the safety of this anesthetic in properly selected cases. That nitrous oxide oxygen is not an efficient anesthetic is frequently indicated by the absence of muscular relaxation and persistence of active reflexes. Attempts to secure satisfactory anesthesia in spite of these elements of inefficiency have resulted in many fatalities, due not to inherent danger from the agent but to abuse of its limited powers. The efficiency of nitrous oxide oxygen can be increased by proper preliminary medication and by the addition of regional anesthesia.

"The efficiency of spinal anesthesia is limited in extent and duration. As with regional anesthesia, the preservation of consciousness is an advantage with some patients and an objection in others. The technic is simple. Muscular relaxation is the most perfect attained by any anesthetic, so that abdominal operations may be done more quickly, compensating to a considerable extent for the inefficiency of the method as to duration. When the time limit of spinal anesthesia is overstepped by the length of the operation, a general anesthetic is used to prolong anesthesia. It is questionable whether general anesthesia throughout, supplemented by regional anesthesia to secure relaxation, would not be preferable. Statistics show remarkable gains in the safety of spinal anesthesia in recent years, doubtless due to the introduction of the less toxic agent, novocaine or neocaine. It is still possible to find reports of one death to 100 cases.

"The hypnotic drugs, in over dosage, produce pronounced respiratory depression and are not suitable for production of complete anesthesia. For preliminary preparation for general or regional anesthesia, they are of great value.

"For operations about the face and upper air passage insufflation is invaluable.

"In obstetrics, the anesthetic must be carefully chosen. Respiratory depression of the new born child and additional burden of the already overtaxed kidneys of the mother must be avoided."

The complete bibliography of 1931 literature would be stupendous. Cited are merely the contributions which your reviewer believes worthy of special mention.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18401

MYOCARDIAL FAILURE OBSCURING THE UNDERLYING LESION

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. ALFRED KRANES*: This is the case of a fifty-nine year old white married American machine operator who came into the hospital through the Emergency Ward complaining of dyspnea, orthopnea and some edema of about three weeks' duration.

The illness started twenty months before entry, when one day while running for a bus he suddenly became very short of breath. He did not think at that time that the exertion was sufficient to cause such a degree of dyspnea. He quieted down however in a few minutes and felt better. Two months after that, eighteen months before admission, while taking a long walk he suddenly became very dyspneic and could hardly catch his breath at all. This dyspnea persisted for some time. When he reached his destination a doctor gave him a hypodermic with marked relief, put him to bed for three weeks and kept him out of work for three months. After that he felt much better. Ever since that time he had always been short of breath on moderate exertion. Three weeks before admission he noticed marked increase in dyspnea and began to be very orthopneic, needing four or five pillows where he had used one before. He had to sit on the edge of the bed a great part of the night trying to catch his breath. At times when he did fall asleep he would be awakened by sudden dyspnea which would gradually wear off. As a result of the loss of sleep he had become very irritable and could not get along well at home. A cough started two or three weeks before admission. He raised some whitish sputum sometimes described as foul. His wife, however, did not think it was really foul. Occasionally there were streaks of blood in it. One other thing that bothered him was a sense of pressure in the precordium or the left hypochondrium. This did not come on at any particular time of day and was not related to emotional excitement; it occurred at any time. It was usually relieved by belching gas or by some counterirritant such as musterole. A week before admission he had edema of the ankles for the first time.

The past history is not significant except for the fact that at sixteen he had a stiffness of both knees lasting two months. A doctor called it growing pain. The patient had two attacks of pneumonia as a child and influenza in 1918.

The family history is interesting in that his father died of heart trouble, his mother of hardening of the arteries.

When he was first seen in the Emergency Ward he was sitting up in bed, moderately dyspneic and somewhat cyanotic. The heart was quite enlarged to the left. No thrills were felt either at the apex or the base. The sounds were of rather poor quality. Over the apex there was a soft blowing systolic murmur and over the base, particularly in the aortic area in the second right interspace, a harsh roughened systolic murmur transmitted up into the neck. The aortic second sound was louder than the pulmonic second. The blood pressure at that time was 150/115. There were râles at both bases. It was difficult to tell whether the liver was down or not because he could not lie flat and was so extremely dyspneic that it was not wise to disturb him. There was pitting edema of both ankles, nothing very marked.

When he was examined the next morning he had the most marked Cheyne-Stokes respiration I have ever seen. He had periods of apnea lasting for thirty seconds. The heart at that time showed protodiastolic gallop rhythm. There were no other significant changes in the heart signs.

The urine on admission had a slight trace of albumin, a few white cells and occasional red cells, many hyalin and granular casts. The white blood cell count was 7,000, the red count a little over 4 million, the hemoglobin 70 per cent, the differential count normal. The Hinton was negative. The non-protein nitrogen was 57 milligrams.

During the course of the day after admission the temperature went up to 103.5°. He complained of some pain in the right axilla. There was dullness at the right base posteriorly with bronchial breathing and many fine râles. The blood pressure dropped to 100/80. Later that day and the next day the temperature went up to 104.3° and finally persisted. He gradually grew worse. He was very hard to control. He was a rather excitable man, and was very much excited after the first dose of morphine. Scopolamin quieted him temporarily. He was moderately cyanotic. He tried to get out of bed. He failed rapidly, and died on the third day.

CLINICAL DISCUSSION

DR. GERALD BLAKE: This man had apparently weathered a mild decompensation two years before, but during the year and a half before he

*Recently resident on the Children's Medical service.

came in he was on digitalis and had got along fairly well, with some symptoms of myocardial weakness. At the time of admission to the hospital he was quite ill, yet although there was congestive failure the diastolic pressure was still 115. No thrill was felt. There were signs at the bases of the lungs following the rise in temperature, and there was pain consistent with the pain of pneumonia.

DR. PAUL D. WHITE: You found no thrill, but very loud murmurs?

DR. BLAKE: Yes.

DR. WHITE: The low blood pressure could be explained by heart failure, by old aortic stenosis complicating hypertensive heart disease, shown by that first blood pressure reading when the diastolic pressure was 115. Aortic stenosis with or without hypertension may give rise to failure with a story like this, but one would have to think also of coronary disease complicating hypertension.

DR. WILLIAM B. BREED: I am impressed with the story of insomnia in the absence of orthopnea as such, occurring as it did here before the onset of congestive failure. I wonder whether that might not be a manifestation of bad coronaries. The question whether he had a good deal of coronary sclerosis seems to me rather an important one. One occasionally gets a story of insomnia in people who later develop infarcts of the heart. I merely bring it up as a distinct possibility that this man's coronaries may have been considerably sclerosed and that he may have had an infarct at the end. What do you think about that, Dr. White?

DR. WHITE: I am much interested in the insomnia because last night I saw a patient whose chief complaint was insomnia following four days of coronary thrombosis. He also had dyspnea. He was an extremely nervous man. I thought that his insomnia was the result of worry about his condition plus rather constant dyspnea. He had been lying rather flat in bed and was decidedly uncomfortable, without pain but with some dyspnea. The respiratory embarrassment plus worry I think accounted for the insomnia. I do think that insomnia of this sort is an early symptom of heart failure sometimes.

DR. BREED: I was struck with this symptom first in the case of a friend of mine who during all one winter complained entirely of insomnia, nothing else. Towards spring he died very suddenly of massive infarct of the heart. Since then I have been interested in the relation of insomnia to coronary sclerosis. Whether it is a symptom of early congestive failure or whether it is an accompaniment of coronary sclerosis is unexplained.

DR. WHITE: I do not believe it is simply reflex from coronary sclerosis. There has to be something else, a very nervous factor or actual symptoms of discomfort like dyspnea.

DR. WILLIAM D. SMITH: I should like to ask about the use of chloral in this case. I note that morphia did not do him much good, but rather excited him. I remember that Dr. Shattuck used to feel that chloral hydrate was a safe drug to give this type of patient. I should like to ask Dr. White if chloral is generally regarded as a dangerous drug to give a patient in this condition?

DR. WHITE: I do not believe so. We are perfectly justified in using one drug after another in order to get relief, morphia or whatever other narcotics are effective. There is no reason why chloral hydrate should not be given so far as I can see. As a matter of fact this patient I spoke of had an excellent night after some luminal plus digitalis. Digitalis may or may not have had a part in the effect. He had six-tenths of a gram.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Arteriosclerotic heart disease.
Congestive failure.
Bronchopneumonia.

ANATOMIC DIAGNOSIS

Rheumatic heart disease; aortitis; aortic stenosis with calcification.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: Autopsy on this patient showed an extremely large heart which weighed 870 grams and a very marked calcified aortic stenosis of the senile type. He also had a most interesting aorta. There were several points of rather marked intimal thickening of the fibrous type without any atheromatous deposits. This thickening was quite irregular on its surface, with a great many very minute elevations that almost suggested vegetations of a type one sometimes sees on the auricular wall when endocarditis extends upward from the mitral valve into the auricle. There was a good deal of the so-called "tree-barky" longitudinal depression, but there were no stellate scars. At the time of autopsy we were in considerable doubt on the basis of the gross appearance whether we should call it a luetic or a rheumatic aortitis. The cases of rheumatic aortitis that we have definitely proved here during the past year have convinced me that we have been missing a very large proportion of those cases before, and that quite a number of cases that in the past we have without hesitation called luetic are in all probability rheumatic. Calcified aortic stenosis does not go with luetic heart disease of course, whereas it does go with rheumatic. We have a questionable history of rheumatic fever in the past in this case. We have no history of lues, and the Wassermann was negative.

The microscopic sections of the aorta show areas of extremely marked intimal thickening.

With the low power it looks rather like atheroma, but when one comes down with the higher power one finds no crystals or vacuolated mononuclears present as in atheromatous plaques of that size, but instead a zone of edematous separation of the collagen fibrils. When we get out into the adventitia we have large numbers of lymphocytes running along the outer border, not particularly perivascular in distribution. The media shows a well marked increase in vascularity with a few arterioles running well into the media and a particularly large number of dilated capillaries around which are numerous lymphocytes. Differential stains show a diffuse increase in collagen, no destruction of elastic fibrils and a marked diminution in muscle cells I am not sure how reliable a differential point that is between luetic and rheumatic aortitis, though it seems to hold very well in the cases I have studied.

DR. WHITE: Was there much fat in the intima?

DR. MALLORY: There was very little actual fat there. So that I think there is every probability that this represents an old rheumatic aortitis and that the aortic stenosis also was in all probability rheumatic in origin.

DR. WHITE: What about the cells in the adventitia? Does that mean an active process?

DR. MALLORY: Not necessarily. Once lymphocytes come into an area where there has been inflammation they may persist almost indefinitely.

DR. WHITE: Was there any proof of a recurrent rheumatic attack?

DR. MALLORY: I hardly think we can say so. I do not think the process looks wholly quiescent, but I think it is largely so. We found a few lymphocytes around some of the blood vessels in the myocardium, but nothing that could be classified as monocytes.

I think it is another case where if it had been possible to make the proper x-ray studies we might perhaps have been able to see the calcified valve.

DR. AUBREY O. HAMPTON: I am sure we could have seen it by fluoroscopy, and if the film were taken fast enough, on a film too. Calcified valves have been very well demonstrated on the film.

DR. WHITE: Are you beginning to pick them up more and more?

DR. HAMPTON: I think that if we look especially for them we can see them very easily.

DR. MALLORY: I should think perhaps the signs were suggestive enough in this case to have made it worth while to establish a diagnosis by x-ray. I do not believe it could have been established in any other way.

CASE 18402

TWO RAPIDLY PROGRESSIVE CASES OF ANEMIA IN ONE FAMILY

CHILDREN'S MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. WILLIAM M. GAMMON*: A ten year old white boy was admitted to the hospital on March 26 because many pinpoint purpuric spots had appeared over the body.

Three years before entry he was admitted to the Eye and Ear Infirmary for a right chronic dacrocystitis. Following operation he was discharged as well. He had had measles, whooping cough and mumps. He was apparently a normal boy until the onset of the present illness.

A brother eight years old had died in this hospital the previous year with a diagnosis of aplastic anemia. The parents and four younger children, three girls and one boy, were well.

A month before admission the patient noticed a purple spot the size of a dime on his left hip, not accounted for by injury. For two days before admission he had had a cold with no general symptoms. The day before admission his mother noticed red spots over his body and extremities. There was no history of bleeding.

Physical examination on admission showed numerous petechiae on his body, face and extremities and a few on the mucous membrane of the mouth. There was one decayed tooth. The tonsils were cryptic and moderately red. A soft systolic murmur was heard over the heart. Otherwise the findings on physical examination were normal.

The temperature was 99°, the pulse 100, the respiration 25.

The urine was negative. Blood examination at entrance showed 3,250 white cells, 3.51 million red cells, 32 per cent polynuclears, 4 per cent large lymphocytes, 56 per cent small lymphocytes, 2 per cent mononuclears, 1 per cent eosinophiles, 5 per cent myelocytes, 2500 platelets. The bleeding time was seven minutes, the clotting time one minute and a half. There was poor retraction of the clot after twenty-four hours. A blood culture was negative. Stool examination was negative.

Course in the hospital. The patient became progressively worse. The temperature gradually went up to 105°. There was an increase in the pulse rate. The white cell and red cell counts gradually diminished, as indicated in the table.

Date	Red blood cells	White blood cells	Hemoglobin
March 26	3.51 million	3,250	70 per cent
" 30		3,050	
April 3	2.28 "	2,800	60 " "
" 7	2.09 "	1,000	32 " "
" 11	1.40 "	1,500	26 " "
" 15	1.96 "	1,200	
" 19	1.26 "	650	28 " "
" 20	0.98 "	250	

*Interne on the Children's Medical service.

There were reticulocytes seen and after the entrance examination the platelets were practically absent from the blood smear.

X-ray examination of the long bones was negative.

On April 4 a biopsy from the sternum was done. The nose and gums began to bleed. Transfusions were done on three different occasions, April 5, 11 and 19. On April 20, twenty-six days after the onset of symptoms, he died.

The history of the brother of this patient is as follows.

The illness began in March, one month before admission. The onset was with the appearance of purplish spots from the size of a pinhead to that of a dime, mostly of the extremities. He complained of weakness and was put to bed. Pallor became marked. Four days before admission to the hospital he had a severe nosebleed followed by a large tarry stool. He was sent in by his physician with a question of acute endocarditis.

His blood picture showed 4,500 white cells, 0.56 million red cells, hemoglobin 15 per cent. The smear showed 60 per cent polynuclears, 34 per cent lymphocytes, besides 6 per cent large mononuclears. The platelets were very few. There were less than one per cent reticulocytes. The bleeding time was twenty-two minutes, the clotting time four minutes.

A systolic murmur was heard over the precordium.

The temperature on admission was 98.6°, but it shortly rose and for the ten days that he was in the hospital subsequently it was between 103° and 106°.

Five transfusions were given, his red cell count rising as high as 3 million on one occasion. The white cell count fell to between 1,000 and 1,800. Bleeding from the nose occurred several times. The child developed mumps from an exposure received before entering the hospital. He died on May 7, about six weeks after the onset of the illness and two weeks after admission to the hospital.

On account of the restricted examination no anatomic diagnosis was possible in this case.

Clinical Discussion

Dr. Harold L. Higgins: The histories of these two boys are very similar. One cannot help but feel that the etiology was the same in both cases. As to what the etiology was, we are very much in the dark. There is nothing that was found on examination of the children that gives us a clue. We inquired quite carefully into the question whether perhaps there might have been some benzol used in the home and whether the illnesses were manifestations of benzol poisoning. The second child was brought in immediately. We had some feeling that the first child was brought to us too late, but we were able to do no better in our therapy with the second child than with the first. The first child was sent in with a diagnosis of acute endocarditis, made apparently on the basis of a heart murmur which probably was a functional or hemic murmur. There was considerable fever in both cases, but we never got any indication that that fever was caused by bacterial toxins. Our diagnosis in both of these cases was aplastic anemia. What caused the aplasia we do not know.

We wonder as to the prognosis of the third brother in the family, who is now six years old.

Dr. Richard C. Tefft, Jr.: The only thing I can add is that the spots that he had were not at all the ordinary spots we find in purpura. They were definite petechiae. We considered ordinary purpura as a remote possibility, but his course was not that of purpura. We considered subacute bacterial endocarditis on account of the many petechiae he had and their definite character, but we could not make out anything that closely resembled subacute bacterial endocarditis. Blood cultures were negative, and the course was a little too rapid for that diagnosis.

There did not seem to be anything we could do. We gave several transfusions in the hope that he might show some improvement, but without feeling that they would do much good, and they did not.

Clinical diagnosis (from hospital record)

Aplastic anemia.

Anatomic diagnoses

Anemia, aplastic.
Purpura.
Acute fibrinous pleuritis.
Hydrothorax, left.
Pulmonary congestion and edema.
Lymphoid hyperplasia.
Atrophy of the thyroid gland.
Multiple hemorrhages in the lungs, myocardium, pericardium and both middle ears.
Septicemia, hemolytic streptococcus and staphylococcus aureus.

Pathologic Discussion

Dr. Tracy B. Mallory: The gross pathology at autopsy showed very little of significance in this case except for the presence of innumerable hemorrhages everywhere. In the lungs there were so many of them and the hemorrhage was so intense that actual hemorrhagic consolidation had been produced and the picture was very close to that of infarction. The bronchial lymph nodes were greatly enlarged, some of the other

lymph nodes moderately so. Microscopic examination however shows nothing but an endothelial and reticular hyperplasia in the lymph nodes. They do not seem to be taking any very essential part in the picture.

We took marrow specimens from a great many of his bones. Those from the sternum show a large proportion of fat cells, perhaps slightly though not much above normal; but in between the fat cells we have many fewer marrow elements than we should have. All types of blood cells are apparently being formed; red cells, white cells, eosinophiles and even a few megakaryocytes are present, but all of them in much diminished numbers as compared with those found in a normal child's marrow.

The specimens from the other bones present much the same picture. Those from the spine and ribs show perhaps a little more function than those from the sternum, but not a great deal. Sections from the long bones, the femur and the tibia, show a very slight marrow function indeed. The exact age at which the long bones cease to function as hematopoietic organs I do not know. I should imagine that at his age, ten years, there would be very little marrow function left in them. But at least we have no compensatory hyperplasia there.

The sections from his liver and spleen show nothing suggestive of leukemic infiltration.

This case one would have to classify on the basis of the facts we have as aplastic anemia. I think it is very interesting however in this connection to recall a case presented last spring, a man named C. who also showed at autopsy an aplastic marrow but who happened to have a bone marrow biopsy done seventeen days before death which showed the characteristic picture of leukemia. That case of C. raised in my mind the question whether we are able to tell at autopsy the difference between aleukemic leukemia and aplastic anemia with any certainty. If we had had only the autopsy findings we should certainly have called C. an aplastic anemia, yet the bone marrow biopsy was typical of leukemia. It raises the question whether a significant percentage of cases which show aplastic anemia may not really be cases of leukemia in which for some unknown reason near the end of the battle the abnormal marrow elements disappear entirely, leaving cell-free marrow. The only possible way of proving such a theory would be to do bone marrow biopsies very early in these cases of apparent aplastic anemia. It would be a very interesting thing to do if it could be done without prejudice to the patient. I believe we did have a biopsy in this case.

DR. HIGGINS: Yes. It was done April 4 and he did not die until the twentieth. The biopsy was done about ten days after the onset of his symptoms.

DR. MALLORY: It does not fit in with my theory, does it?

DR. HIGGINS: It showed questionable aplastic anemia.

A PHYSICIAN: In this case of C. with the biopsy and with aplasia of the bone marrow was there no infiltration in the liver or spleen?

DR. MALLORY: That had all disappeared. On the other hand typical leukemias at least occasionally come to autopsy with no infiltration of the liver. A case of Dr. Urmy's, Ch., had no infiltration of the liver at autopsy, though it was otherwise as typical a leukemia as one could ask for.

A PHYSICIAN: Was there any x-ray treatment after the biopsy in the case of C.?

DR. MALLORY: There was no x-ray treatment at all.

DR. ALLAN G. BRAILEY: As I remember it that patient C. had been exposed to some arsenic spray. Is there any possibility of that in the case of this boy?

DR. MALLORY: Nothing of the sort was discovered so far as I know, and I believe the local board of health investigated the cases fairly carefully.

DR. HIGGINS: You found nothing bacteriologically to explain it?

DR. MALLORY: There was a hemolytic streptococcus in the heart's blood, but I think that in all probability that was a terminal event only.

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PULMONARY EMBOLISM

THE dramatic qualities of death from pulmonary embolism have naturally kept this tragic complication of operative procedures a frequent subject of discussion. Much has been written on the prevalence of the condition. Statistics can be found proving that it is increasing, that it is decreasing, and various procedures either reduce its incidence or are without effect. Various theories of etiology have been compounded based on an assumed increase in the incidence of the disease. Anesthesia has borne its share of the blame but over large series there is no significant difference between the incidence of fatal pulmonary embolism in general anesthetics of different types or spinal or local anesthesia.

The most valuable recent contribution to this subject is the review by Rosenthal[1]. He has gathered together the statistics from twelve general clinics of Central Europe, giving the percentage incidence of both thrombosis and fatal pulmonary embolism. In general these clinics showed a slight increase in the incidence of these conditions from 1910 to 1914, practically no change through the period of the war, and after the war a steady increase, reaching its peak about 1928. Paralleling these figures he shows those from ten surgical clinics, which again show an increase after the war, reaching a peak in 1927. In general this increase is much more marked in the surgical clinics than in the general clinics. In 1928 a slight decline in the incidence of fatal pulmonary embolism began in the surgical clinics. This, however, has not progressed to an appreciable extent. These figures are compared with those of eleven surgical clinics in this country, which show remarkably little change over a period of years, although of course marked variation from year to year. There is no such increase shown here as was encountered in the European clinics.

Rosenthal believes one of the causes of the increase in thrombosis and embolism to be the increased prevalence of cardiovascular disease, particularly manifest during the inflation period in Central Europe.

REFERENCE

1 Rosenthal, S. R.: Thrombosis and fatal pulmonary embolism. Arch. Path. 14:215 (Aug.) 1932.

MASTER JOHANN DIETZ

THE Medieval Guild of Barber Surgeons persisted on the continent of Europe even into the early eighteenth century and formed the link of continuity between the primitive surgery of the earliest times and that of our own epoch. Our knowledge of these early barber surgeons and their activities is derived from many sources, one of the most valuable of which, particularly with reference to the latest days of the Guild, is the autobiography of Johann Dietz, surgeon in the Army of the great Elector, Friedrich Wilhelm (1640-1688), and barber to the Royal Court of Brandenburg. The manuscript of this autobiography, composed during the author's old age, lay for years unpublished in the Royal Library of Berlin; but it was finally translated by Bernard Miall and first published in English by Dr. Ernst Consentius in 1923. For any modern surgeon interested in the history of his craft, this accessible volume deserves perusal not only for its amiable style of narrative but for the vivid picture which it affords of the homely middle class life of the barber surgeon in his palmy days in Germany of the latter seventeenth and early eighteenth centuries.

The early chapters describe Johann's parentage and education. He was the son of a rope maker in Halle and his father was aggrieved because his promising son was unwilling to follow the paternal occupation but was ambitious to become a barber surgeon like his mother's brother, Master Georg Schobern. The boy, however, had his way and was indentured to his

uncle in 1681. He describes amusingly the experiences of his apprenticeship and presents a vivid picture of the pestilence, presumably Bubonic Plague, which decimated the population of the town during this period. He, himself, employed in opening buboes on patients, acquired the infection and was shut up by his father in a barn to die. Happily he recovered after taking a plague essence which he had himself concocted but of which he does not give the formula.

More than half of the text of Dietz's autobiography is concerned with the travels which he pursued as a journeyman barber during the years following his apprenticeship. He served both in the Army and in the Navy and in civilian capacities, and his journeys took him over a large part of Europe. As army surgeon he took part in the Brandenburger's campaign in Hungary when the Fortress of Ofen was captured; and in the capacity of chief surgeon he made a voyage with the whaling fleet into the Arctic. Later in Berlin he was assistant to the celebrated Andreas Horch who was regimental surgeon to the Brandenburg electoral footguards and a figure of great celebrity in his day. Dietz's discursive and prolix narrative is fascinating for its naïveté and vividness of detail, for the sidelights which it affords on the medical and surgical practices of his time, and for the ingenuousness with which it mixes intimate personal details with the main thread of the story.

From time to time during and after his travels, Dietz returned to his native Halle. Here he was twice married and by his second wife had four children when he was over seventy. It was during his period of retirement, after he was seventy years of age, that his autobiography was composed as the somewhat garrulous but delightful reminiscences of an old barber surgeon with a wealth of picturesque experience in life, living on into the early days of the more modern surgery which he, himself, viewed with a certain amount of playful skepticism.

Master Johann Dietz's manuscript, then, is in the history of medicine, a document of extreme value, summing up the history of the craft which he represented so typically in his personal experience. It is a source which materially enriches our knowledge and affords us, perhaps the best picture which we have of the professional and civilian life of the time when the great Elector was still reigning, when the magnificent Friedrich First kept his brilliant Court, and the niggardly Friedrich Wilhelm First was organizing his austere government. It is a document which deserves to rank, though in a more limited field, with the immortal diaries of Evelyn, Pepys, and Sewall.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

FRIED, B. M. M.D. Imperial Nicholas University Medical School, Saratow (Russia) 1914. Assistant in Medicine, Beth Israel Hospital, Boston. His subject is "Primary Chronic Splenomegaly. Banti's Disease." Page 595. Address: 145 W. 85th Street, New York City.

SWAN, CHANNING S. A.B., M.D. Harvard University Medical School 1923. F.A.C.S. Assistant Urologist, Massachusetts General Hospital. His subject is "Results and Interpretation of a Complement Fixation Test for Gonorrhea." Page 601. Address: 264 Beacon Street, Boston.

NEIFELD, H. B.S., M.D. Medical College of Virginia 1924. Member of the American Editors and Authors Association, Kings County Section of Physio-Therapy Society. His subject is "Diathermy Treatment of Acquired Balanitis, Phimosis and Paraphimosis." Page 603. Address: 167 Willoughby Street, Brooklyn, New York.

KIERNAN, FRANK. A.B. Executive Secretary, Massachusetts Tuberculosis League. His subject is "Health Education Demonstrations in Massachusetts High Schools." Page 604. Address: 1148 Little Building, Boston.

BURPEE, BENJAMIN P. A.B., M.D. Harvard University Medical School 1914. Obstetrician, Elliot Hospital, Manchester, N. H. His subject is "The Conduct of Normal Labor." Page 607. Address: Manchester, New Hampshire.

CLOW, FRED ELLSWORTH. M.D. Harvard University Medical School 1904. Visiting Physician, Huggins Hospital, Wolfeboro, New Hampshire. His subject is "Anorexia Nervosa." Page 613. Address: Brown House, Wolfeboro, New Hampshire.

SHELDON, R. F. A.B., M.D. Harvard University Medical School 1911. Assistant Anesthetist, Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary. His subject is "Progress in Anesthesia in 1931." Page 620. Address: 31 Pinckney Street, Boston.

MISCELLANY

NOMINATION OF DR. MARK SHRUM

Governor Ely has nominated Dr. Mark Shrum to membership on the Board of Registration in Medicine. Dr. Shrum was born in 1869 and is a graduate of the University of Louisville School of Medicine. This nomination will be considered by the Governor's Council at its next meeting.

CHARGES AGAINST TWO BOSTON DOCTORS ARE REFERRED TO THE DISTRICT ATTORNEY OF SUFFOLK COUNTY

Indictments in Middlesex County against two Boston physicians have been turned over to the District Attorney of Suffolk County because of lack of authority in the Middlesex Courts.

The indictment alleged conspiracy to defraud insurance companies. Both of the physicians whose names have been published in a Boston newspaper are graduates of Class A medical schools and have standing among their associates.

It is explained that the alleged conspiracy originated in Boston and hence further action, if any, should come under the jurisdiction of Suffolk County. Both of the doctors have pleaded not guilty. It will be a relief if further action will establish their innocence.

CORRESPONDENCE

THE RESTORATION OF THE LICENSE OF GEORGE WARREN EASTMENT

The Commonwealth of Massachusetts
Department of Civil Service and Registration
Board of Registration in Medicine
State House, Boston
September 28, 1932.

The *New England Journal of Medicine*,

At a special meeting of September 23, 1932, the Board of Registration in Medicine voted to restore the license of Dr. George Warren Eastment, of 56 Central Avenue, Lynn, Massachusetts, which had been cancelled on August 20, 1931.

Very truly yours,
STEPHEN RUSHMORE, M.D., *Secretary*.

RECENT DEATHS

THURBER — DR. MADISON TEMPLETON THURBER died at his home, 30 Thornley Street, Dorchester, Mass., September 27, 1932. Dr. Thurber was born in Webster, New Hampshire, in 1866. His early education was acquired in the public schools of Webster and Manchester. He fitted for college at the Military Academy in Pennacook and the New Hampshire Agricultural and Mechanics Arts School at Hanover, and took his M.D. degree from Dartmouth in 1890. He practiced in Grafton, New Hampshire, for four years preceding his removal to Boston. He was the organizer and first President of the Dorchester Medical Society and was a member of the State Medical Societies of Massachusetts and New Hampshire.

He served as Medical Examiner of the New Hampshire Mutual Life Insurance Company and was visiting gynecologist at the Boston Emergency Hospital for several years. He was prominent in civic affairs having held membership in the Mt. Cardigan Knights of Pythias Lodge, the Union Lodge, A. F. & A. M., the Episcopal Club of Massachusetts and Boston Council, R. & F. M. M. He was a communicant of the Church of the Advent.

Two children survive him, Madeline W. Thurber and Lincoln Joseph Thurber.

CHADWELL—DR. ORVILLE ROGERS CHADWELL died suddenly at his home, 793 Centre Street, Jamaica Plain, September 30, 1932. His Boston office was at 510 Commonwealth Avenue. Dr. Chadwell was born in Lynn, Massachusetts, August 26, 1877. He was graduated from the Boston University School of Medicine in 1903.

He had served on the teaching staff of Boston University School of Medicine in the department of pediatrics and was a member of the Staff of the Massachusetts Memorial Hospitals.

He joined the Massachusetts Medical Society in 1917.

He is survived by a widow and a daughter.

NOTICES

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The next clinic will be given on October 13.

Also physicians and medical students are invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays.

RADIO HEALTH MESSAGES

OCTOBER-NOVEMBER-DECEMBER, 1932

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:15 P.M.

October
11 What the State Is Doing for Crippled Children
18 Old Age Deferred
25 Some Facts Worth Knowing about Your Heart

November
1 Tuberculosis—Prevent It
8 Our Vitamins
15 The Health Examination
22 Only a Sore Throat
29 Anemia or the Lack of Blood

December
6 Sickness and Character
13 How Are You?
20 Chorea or St. Vitus' Dance
27 Hemorrhoids

REPORTS AND NOTICES OF MEETINGS

WACHUSETT MEDICAL SOCIETY

The annual fall meeting of the Wachusett Medical Society was held in Holden District Hospital on September 21. The officers elected for the ensuing year are President Dr. O. Draper Phelps; Vice President, Dr. Ernest B. Emerson; Secretary-Treasurer, Dr. Robert J. Northridge.

At the scientific meeting Dr. Joel Melick read a paper on "Repair of Both Old and New Perineal Lacerations Immediately Following Delivery." The talk was illustrated by moving picture films of operations performed by the author.

ROBERT J. NORTHRIDGE, M.D., *Secretary.*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held Tuesday, October 18, 1932, 12 noon, at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

The speaker will be Robert B. Osgood, M.D., and his subject, "'Public Sickness' as a Means of Improving Public Health and of Conserving Private Practice in Chronic Rheumatism."

All physicians are cordially invited to attend. There will be the usual luncheon at 1 o'clock.

AN INVITATION TO PHYSICIANS

SUFFOLK DISTRICT MEDICAL SOCIETY AND THE BOSTON MEDICAL LIBRARY

Dear Doctor:

You are cordially invited to attend the five monthly meetings of the Suffolk District Medical Society and the Boston Medical Library to be held during 1932-33 as specified below on Wednesdays at 8:15 P.M.

October 26, 1932
Stated Meeting
Clinical Program, Boston City Hospital:
Initiative Petition of Chiropractors. Dr. Thomas J. O'Brien.
Certain Atypical Manifestations of Subacute Bacterial Endocarditis. Demonstration. Dr. Soma Weiss.
Cardiac Hypertrophy. Dr. F. B. Mallory.
Treatment of Tetanus. Dr. William R. Morrison.
New Growths of Large Bowel. Dr. Arthur R. Kimpton.
Clinical Demonstration of Cases. Dr. Ralph C. Larrabee.
Comparative Mortality Rates of Acute Appendicitis. Dr. Irving J. Walker.

November 30, 1932
Clinical Program, Peter Bent Brigham Hospital

January 25, 1933
General Meeting in association with the Boston Medical Library
Titles and speakers to be announced later

March 29, 1933
Clinical Program, Massachusetts General Hospital

April 26, 1933
Annual Meeting
Election of officers
Scientific program—Titles and speakers to be announced later

The Medical Profession is cordially invited to attend all of these meetings.

WILLIAM H. ROBEY, M.D., *President,*
LELAND S. MCKITTRICK, M.D., *Secretary,*
HILBERT F. DAY, M.D., Boston Medical Library.

COMBINED MEETING OF THE PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE, PHILADELPHIA PEDIATRIC SOCIETY AND NEW ENGLAND PEDIATRIC SOCIETY

SATURDAY, OCTOBER FIFTEENTH, NINETEEN HUNDRED AND THIRTY-TWO, BOSTON, MASSACHUSETTS

MORNING SCHEDULE

Members arriving from New York and Philadelphia should get off the train at Back Bay station and go directly to the Harvard Club, 374 Commonwealth Avenue, where breakfast will be served from 7:30 to 9:00 o'clock.

Harvard Medical School, Amphitheatre D

10:00 A.M. An Interpretation of the Functions of the Sympathetic Nervous System. Dr. Walter B. Cannon.

10:30 A.M. Some Aspects of Secondary Anemia. Dr. George R. Minot.

11:00 A.M. Basal Metabolism Determinations in Children: Their Clinical Significance. Dr. Fritz B. Talbot.

11:30 A.M. What Is Celiac Disease? Dr. John L. Morse.

12:00 M. What Is Nephrosis? Dr. Henry A. Christian.

12:45 P.M. Luncheon at Longwood Towers, Brookline.

AFTERNOON SCHEDULE

Children's Hospital Amphitheatre, Longwood Avenue

3:00 P.M. Vitamin A Deficiency. Dr. Kenneth D. Blackfan.

3:30 P.M. Demonstration of Patients with Endocrine Dysfunction. Dr. Gerald Hoeffel.

4:00 P.M. Considerations on the Use of Protein and Salt in the Treatment of Various Types of Bright's Disease. Dr. Allan M. Butler.

4:20 P.M. Streptococcus Tracheobronchitis. Dr. Lyman Richards.

4:40 P.M. The Relation of Anemia of the Newborn to Familial Icterus Gravis and Erythroblastosis. Dr. Louis K. Diamond.

5:30 P.M. Refreshments at Harvard Club. (Aesculapian Room.)

7:30 P.M. Dinner at the Hotel Kenmore, 490 Commonwealth Avenue.
Dr. George H. Bigelow, Health Commissioner of Massachusetts—The Relationship of Public Health Activities to the Practice of Pediatrics.

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New York Academy of Medicine

Chairman: Dr. John Caffey, 50 Haven Avenue, New York, N. Y.

Secretary: Dr. Harry Bakwin, 132 East 71st Street, New York, N. Y.

Philadelphia Pediatric Society

President: Dr. John P. Scott, 2040 Pine Street, Philadelphia, Pa.

Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Avenue, Bala-Cynwyd, Pa.

New England Pediatric Society

President: Dr. Lewis Webb Hill, 319 Longwood Avenue, Boston, Mass.

Secretary: Dr. Gerald Hoeffel, 319 Longwood Avnue, Boston, Mass.

AMERICAN ACADEMY OF PHYSICAL THERAPY

PROGRAM
TENTH ANNUAL MEETING

October 12, 13 and 14
Hotel Walton, Philadelphia

WEDNESDAY MORNING, OCTOBER 12, 9 O'CLOCK

Business Session.

Scientific Session opens at 10 o'clock.

1. Mineral Water, an Appraisal.
   Miletus Brown Jarman, Hot Springs, Virginia.
2. The Sun-Climate of Miami, Florida.
   Otto J. Seiplein, Ph.D., Miami, Florida.
3. Effect of Change in Climate in the Rheumatic Infection in Children with Heart Disease. (With lantern slides.)
   T. Duckett Jones, M.D., Boston, Massachusetts.

   Discussion of this group of papers by:
   Joseph H. Adams, Sc.D., Miami Beach, Florida.
   C. F. Roche, M.D., Miami Beach, Florida.
   William H. Watters, M.D., Miami, Florida, and Boston, Mass.

Address, 12 o'clock noon

4. Physical Therapy in Neurology.
   Theodore H. Weisenburg, M.D., Philadelphia.

AFTERNOON, CLINICS AT 2 O'CLOCK

Clinics will be held each afternoon at one of the following hospitals:

Jefferson Medical College Hospital—Dr. William H. Schmidt.

Temple University Hospital—Dr. Frank H. Krusen.

Graduate Hospital of the University of Pennsylvania—Dr. William T. Johnson, Dr. Frederick A. Cochran.

Hospital of the University of Pennsylvania—Dr. Josef B. Nylin.

(See bulletin board for schedule.)

WEDNESDAY EVENING, 7 O'CLOCK

Round Table Dinner.

Informal discussion will be led by Dr. R. Tait McKenzie, Dr. Ralph Pemberton and Dr. William L. Clark.

THURSDAY MORNING, OCTOBER 13, 9 O'CLOCK

1. Diathermy.
   Frank H. Krusen, M.D., Philadelphia.
   Discussion by A. Bern Hirsh, M.D., New York City.
2. The Static Wave Current, Its Uses in Some Common Conditions.
   William Martin, M.D., Atlantic City, New Jersey.
   Discussion by William D. McFee, M.D., Boston, Massachusetts.
3. Value of Rapid Sinusoidal Current in Sciatica and Similar Neuromuscular Conditions.
   Benjamin Ulanski, M.D., Philadelphia.
   Discussion by William H. Schmidt, M.D., Philadelphia.
4. Artificial Fever in Chronic Disease with Description of a New Apparatus.
   William H. Schmidt, M.D., Philadelphia.
   Discussion by Byron S. Price, M.D., New York City.

The President's Address
12 O'clock, Noon
William F. Roberts, M.D.,
St. John, New Brunswick, Canada.

THURSDAY AFTERNOON, 2 O'CLOCK

Clinic—See bulletin board for schedule.

FRIDAY MORNING, OCTOBER 14, 9 O'CLOCK

1. Modern Treatment in Mental Disease.
   John A. Holland, M.D., Langhorne, Pennsylvania.
   Discussion by Thomas L. Smyth, M.D., Allentown, Pennsylvania.
2. A New Technique in Muscle Reëducation in Anterior Poliomyelitis. (With motion pictures.)
   Harold D. Corbusier, M.D., Plainfield, New Jersey.
   Discussion by George E. Deering, M.D., Worcester, Massachusetts.
3. Advances in Treatment of Cancer within the Mouth.
   William L. Clark, M.D., Philadelphia.
   Discussion by Edward H. Trowbridge, M.D., Worcester, Massachusetts.
4. Abstract of Dr. Joseph Riviere's Paper on Electrosurgery.
   William D. McFee, M.D., Boston, Massachusetts.

FRIDAY AFTERNOON, 2 O'CLOCK

Clinic—See bulletin board for schedule.

A half-hour recess will be taken each morning for inspection of exhibits.

All members of the medical profession are cordially invited to attend and to participate in the discussion.

ARTHUR H. RING, M.D., *Secretary-Treasurer.*
Arlington, Mass.

WORCESTER DISTRICT MEDICAL SOCIETY

GRAFTON STATE HOSPITAL AUDITORIUM
WEDNESDAY, OCTOBER 12, 1932

6:15 P. M. Buffet Supper. (Complimentary by State Hospital.)

7:15 P. M. Business Meeting and Scientific Program.

SYMPOSIUM

What the General Practitioner Can Do in the Line of Preventive Medicine.

1. "What Laboratory Helps the City and State Health Departments Offer," Dr. Peter O. Shea, Director of Health for the City of Worcester.
2. "Diphtheria," Dr. John C. Ward, Worcester.
3. "Scarlet Fever," Dr. Roy J. Ward, Worcester.
4. "Smallpox," Dr. Oscar A. Dudley, Worcester.
5. "Measles," Dr. Thomas E. Cunningham, Worcester.

The discussion of these papers will be opened by Dr. Gaylord W. Anderson, Deputy Commissioner of the State Department of Health, Boston.

9:00 P. M. Scientific Program to be followed by a less serious talking picture.

The Fall Censors' Meeting will be held at the Worcester Public Library, Elm Street, Worcester, at 4:00 P.M. Thursday, November 3, 1932. Please notify all prospective candidates.

EDWIN R. LEIB, M.D., *President,*
ERWIN C. MILLER, M.D., *Secretary.*

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

October 7—William Harvey Society. See notice on page 594, issue of September 29.

October 12, 13 and 14—American Academy of Physical Therapy. See notice on page 635.

October 13—Clinics in Medicine at the Peter Bent Brigham Hospital. See page 633.

October 15—Combined Meeting of the Pediatric Section of the New York Academy of Medicine, Philadelphia Pediatric Society and New England Pediatric Society. See page 634.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 18—South End Medical Club. See page 634.

October 19—The New England Dermatological Society. See notice on page 557, issue of September 22.

October 19—The New England Physical Therapy Society. First meeting of the season. Detailed notice will appear in a later issue of the Journal.

October 22 and December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice on page 557, issue of September 22.

October 22-24—Health Education Institute. See notice on page 47, issue of July 7.

October 24-27—American Public Health Association. See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

DISTRICT MEDICAL SOCIETIES

HAMPDEN DISTRICT MEDICAL SOCIETY

October 18—See notice on page 594, issue of September 29.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

November 9 at Winchester.
January 11 at Wakefield.
March 15 at Melrose.
May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

SUFFOLK DISTRICT MEDICAL SOCIETY

Joint meetings with the Boston Medical Library. See page 634.

WORCESTER DISTRICT MEDICAL SOCIETY

Schedule of meetings for 1932-33:

Wednesday, October 12—See notice elsewhere on this page.

Thursday, November 3—Fall Censors' meeting. See notice elsewhere on this page.

Wednesday, November 9—Dinner and scientific meeting at the Worcester Hahnemann Hospital. Program to be announced later.

Wednesday, December 14—Meeting at the Worcester Chamber of Commerce Auditorium. Paper by Dr. Joseph Meigs of Boston, entitled "Uterine Bleeding."

Wednesday, January 11—Dinner and meeting at the Worcester City Hospital. Paper by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association.

Wednesday, February 8—Dinner and scientific meeting at the Worcester State Hospital. Program to be announced later.

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEW

Minor Surgery. By FREDERICK CHRISTOPHER. W. B. Saunders Company, Philadelphia. Second Edition. 1932. 998 Pages. Price $10.

The first edition of Christopher's "Minor Surgery" was welcomed as the most valuable contribution to the subject that has yet been made. It has now been thoroughly revised and enlarged although still maintaining the same general scope. The source of material is not limited to his own wide experience, but nearly every page contains quotations or references from the current literature.

New material has been added throughout the book. This is most noticeable in the consideration of vascular diseases, plastic surgery, local anesthesia, especially in fractures, the injection treatment of hemorrhoids, and postoperative care as outlined in the section on instructions to the surgical intern.

Inasmuch as the great majority of surgical cases are those that belong to the field of minor surgery, study of this volume will prove of tremendous value to all who are engaged in surgical practice.